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A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PEDIATRICS, OBSTETRICS, GYNECOLOGY, ORTHOPEDICS,
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,
OTOLOGY, RHINOLOGY, LARYNGOLOGY,
HYGIENE, AND OTHER TOPICS OF INTEREST
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION
THROUGHOUT THE WORLD

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The Summer Diarrheas of Children

MILK BACTERIA AND INTESTINAL DISORDERS

BY H. W. CONN, PH.D.
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It is generally recognized to-day that the bacteria present in milk used as food are responsible for a good share of the intestinal disturbances of summer, particularly those found among children. Various facts which we cannot notice here have led to this belief, and it must be admitted as one of the conclusions in regard to which there is at the present time really no dispute.

But when we attempt to take a step farther in the matter and analyze a little more closely the cause of these intestinal disorders, we are immediately in difficulties; and, in spite of the most thorough light thrown upon the subject by modern study, we still find little besides uncertainty and confusion. The recognition that bacteria are responsible for such diseases has raised the question of whether it is not possible to determine the wholesomeness of milk by bacteriologic study, and has suggested that bacteriologic methods may in time place within the reach of our public health inspectors a means of so controlling the public milk supply as to guarantee its wholesomeness. The bacteriologic study of water has been extremely useful in enabling health authorities to gain a proper control over water supplies. Such study of water, which has now been carried on more and more extensively each year for the last 20 years, has made it possible to reach a tolerably definite conclusion as to whether a given body of water is wholesome and proper for drinking purposes, or whether it is suspicious and should be rejected. The method of bacteriologic study of water has been chiefly a quantitative determination of the number of bacteria present; and while it is recognized that a quantitative study has its limitations, nevertheless much valuable information has been yielded by such work. But at the present time, even in the study of water problems, the question of the number of bacteria is being in a measure supplemented by the more significant question of the species of bacteria. The recognized fact that the presence of Bacillus coli communis is generally an indication of sewage contamination has led our modern sanitarians to the study of the distribution of this particular bacillus in water, and to place fully as much weight upon the determination of this fact as upon the simple determination of numbers. In other words, in the problem of water analysis quantitative bacteriologic study is in a measure being replaced by qualitative study.

The problems presented by the bacteria in milk in relation to its wholesomeness are far more difficult than those of water. It is perfectly evident at the outset that the two problems are totally different; for whereas water that contains a few thousand bacteria per cubic centimeter is usually regarded with suspicion, and in most cases must be regarded as unwholesome, it is demonstrated beyond peradventure that milk may contain bacteria in millions per cubic centimeter, and yet be perfectly wholesome. It is certainly true that much of the milk consumed daily in our cities contains more bacteria than even the worst sewage; and it is equally beyond question that milk with these enormous numbers has been consumed by the human race for generation after generation without apparent injury to the consumer. Undoubtedly milk may contain bacteria by the millions in each cubic centimeter, and yet be perfectly wholesome, while a small fraction of this number of bacteria would usually render water totally unfit to drink. Evidently, the problem of milk bacteria is totally different in its relation to public health from the problem of water bacteria.

But the recognition that milk contains bacteria in such large numbers has been regarded as indicating that the milk is unwholesome. It has been tacitly assumed that the wholesomeness of milk may be, in a measure, determined by the number of bacteria that it contains. In the light of the information we have at the present time, however, it appears that this conclusion is quite unwarranted. Sometimes milk containing large numbers of bacteria is unwholesome; but in many other cases milk with small numbers of bacteria is equally, or even more, unwholesome, and certainly milk containing very large numbers can be, and is, used with perfect impunity.

Clearly enough the problem of the relation of milk bacteria to health must resolve itself into a question of the *kinds* of bacteria rather than the simple question of *numbers*.

What kind of bacteria are concerned in the production of the intestinal disorders in children is not at the present time positively known. It seems that a number of different species of micro-organisms must be concerned therein. The intestinal diseases of children do not by any means constitute a single definite disease. A variety of symptoms arise, and a natural inference is that the causes of the trouble are several. At the present time, unfortunately, we are in ignorance in regard to the actual causes of these troubles. Nevertheless, certain facts already known are at least suggestive. In order to make clear the facts to be referred to, it is necessary to notice briefly the types of bacteria that are commonly found in milk. Scores, and even hundreds, of different species of bacteria have been found in normal milk products, but among this long series of species we may, for convenience of study, recognize three easily distinguishable types.

(I) *The Lactic Bacteria.*—Lactic bacteria are the best known of the types of milk organisms. The characteristic of this group is their power of forming lactic acid from milk sugar, although many other by-products may be formed at the same time. There are many species of bacteria capable of producing this primary action upon the milk sugar. In normal milk the number of varieties of bacteria that produce lactic acid is large,—a score or two of clearly distinct species of micro-organism with this power highly developed being tolerably well known. Among them there is one species, or perhaps better to say a group of species, that stands pre-eminently ahead of all others. This is a type of bacterium the best name for which appears to be Bacterium lactis acidi. It grows better without a free supply of oxygen, produces lactic acid with great rapidity, and causes the typical souring and curdling of the milk in the course of a very few hours. This will be referred to hereafter as the typical lactic bacterium. In addition, there are several other species that are extremely common, and that differ from this in a number of important characters.

(II) *Putrefactive Bacteria.*—A second type of milk organisms is characterized by the power of producing enzymes. In the case of most of these species of bacteria the enzymes are apparently two in number. One is a rennet-like enzyme that precipitates the casein and curdles the milk; the other is a trypsin-like enzyme which digests the casein. These bacteria in general are those which are recognized by bacteriologists as capable of liquefying gelatin. One of the prominent characteristics of this type of organisms, so far as concerns their relation to milk, is that many of them produce putrefaction. If they are allowed to develop in milk they cause a series of decompositions to take place, the result of which is the production of a variety of products with vile odors and smells which we characterize in general by the name of putrefaction. This group of organisms for our purpose may be briefly referred to as the putrefactive type.

(III) *Miscellaneous Bacteria.*—In addition to the two types above mentioned, there is a long series of bacteria which we shall not attempt to classify in any particular group. We shall simply refer to them hereafter as miscellaneous species.

In considering the relation of the different types of bacteria to wholesomeness of milk, we notice first that there is absolutely no reason to believe that the common lactic bacteria are the cause of any trouble to the person who uses milk. The evidence is all against a belief that any form of summer complaint has any connection with the lactic bacteria. The reasons for this cannot be detailed here. It may only be mentioned that sour milk itself is not an unhealthful product, and certainly does not give rise to summer complaint. Sour milk itself contains lactic bacteria in almost incredible quantities, the number being frequently as many as 300,000,000 per cubic centimeter. Butter always contains these same organisms in enormous quantities, and practically all cream that has been kept for a day or two contains them in numbers far beyond those present in the city milk. Cheese, moreover, is filled with lactic bacteria in large quantities. The fact that sour milk, butter, cream, and cheese are recognized as healthful, and certainly as not productive of the series of diseases characterized by intestinal disturbances, is, for our purpose, sufficient to indicate that to the common lactic bacteria we cannot attribute this class of diseases.

It must be recognized, however, that this statement does not necessarily apply to all lactic bacteria. As mentioned, there are many species of lactic bacteria; and it is certainly possible that some among them are sources of trouble. The Bacterium coli, Bacillus typhosus, and the bacterium recently claimed by Duval, Vedder, Bassett, and Hiss, as the cause of summer complaint in children, are organisms which produce acid. None of these are the typical lactic bacterium, Bacterium lactis acidi, to which the above statement applies.

The relation of the putrefactive bacteria to the problem stands differently. While we do not know that the putrefactive bacteria have anything to do with summer complaints, they must certainly be looked upon as suspicious. The putrefaction of food products is known to be accompanied by the production of poisonous materials in the form of ptomains, etc. One of the symptoms connected with a variety of the intestinal troubles is a putrefaction of the intestinal contents. It is quite generally recognized that some types of intestinal troubles are to be regarded as intoxication diseases, *i.e.*, due to the absorption from the intestinal canal of toxic poisons. Now, when we recognize that the general class of putrefactive bacteria certainly do produce poisonous ptomains, and that a putrefaction of the intestinal contents characterizes many of these summer troubles, the conclusion is justified that the putrefactive bacteria must be looked upon as having some share in the development of this class of troubles. It is not meant by this statement to claim that summer complaint is due solely to the putrefactive bacteria, but simply that these general facts suggest to us to look upon the putrefactive microorganisms with suspicion, and to regard the normal lactic milk bacteria as harmless in their action upon mankind. Hence milk that contains large numbers of the putrefactive bacteria should, other things being equal, be regarded with more suspicion than milk that contains these organisms in small numbers.

Not only do the common lactic bacteria fail to show any direct relation to these intestinal disturbances, but they are actually agents for protecting the milk from the growth of other bacteria and guarding it from unwholesome fermentations. To explain this more clearly, a description of the normal growth of bacteria in milk must be given.

When milk is freshly drawn, under common conditions, it is found to contain bacteria in moderately large numbers and in considerable variety; there may be 12, 15, or 20 species present. Of this variety some species may comprise a large per cent., others a small per cent., and others will be present only in small numbers.

In most cases, when this matter has been studied by recent methods, such fresh milk contains only small numbers of the normal lactic bacteria, while the putrefactive forms and the miscellaneous bacteria comprise the large majority; in most cases considerably over 90 per cent. If the bacteria in this same sample of milk are studied hour after hour, we find a rapid change in the proportionate numbers of the different species. Some of these species fail to grow and some disappear; others grow more or less rapidly and hold their own in percentage for a while; while others grow rapidly, increase their percentage, and in the end are in greater proportionate numbers than at the beginning.

The most striking feature of the increase is that the lactic bacteria, comprising at the outset sometimes not more than 1 per cent., not only increase in numbers from the beginning, but rapidly increase in percentage. In successive hours these bacteria will comprise 10 per cent., 20 per cent., 30 per cent. of the whole, and after 24 to 48 hours this particular type of bacterium has so far outdistanced all other species that it frequently comprises from 95 to 100 per cent. of the whole. After milk has become old and about ready to sour, it consists largely of this one species, all other types having been checked or destroyed by the extraordinary development of the lactic bacteria. The conclusion from these facts is that these lactic organisms find milk such a congenial home that they flourish more vigorously than any other species of bacterium, and in the course of time take possession of the milk almost to the exclusion of others. In other words, the lactic bacteria stop the growth of other types, protect the milk from the development of the putrefactive forms, and preserve it from a variety of unpleasant and unwholesome putrefactive fermentations which it certainly would undergo were it not for the growth of the lactic bacteria.

A significant fact was recently brought out by Bienstock in this connection. There is present in the normal intestine a type of bacterium which produces putrefaction, named Bacterium putrificus. Bienstock has found that this putrefying organism does not ordinarily grow if other micro-organisms are present. When there is present in a solution a proper quantity of certain acid-producing micro-organisms, the growth of the putrefying germ is checked, and even apparently prevented, so that the intestinal contents do not putrefy; but if the acid organisms are absent, putrefaction takes place. There is little question that in such cases the reason for the checking action upon the putrefying germs is the development of acid produced by the bacteria. But the presence of this acid is in itself due to the activity of acid organisms, and therefore it is quite intelligible that lactic or other acid germs should prevent putrefaction in the intestine.

From all of these facts, it is evident that a totally different interpretation must be placed upon the presence of lactic bacteria in milk and the presence of other types. The lactic bacteria produce a quantity of lactic acid causing the milk to sour and eventually to curdle, but apparently they are harmless, and may be present in enormous numbers without rendering milk in the slightest degree unwholesome. Inasmuch as they have the power of checking the development of other organisms, they may actually, at least under some circumstances, render the milk more wholesome by preventing the development of the types of micro-organisms which would be likely to produce trouble. Milk that contains large numbers of such lactic bacteria need not be looked upon as unwholesome; whereas, milk that contains a very much smaller number of other kinds of putrefying bacteria is at least suspicious.

It is certainly desirable to have the bacteria in milk as few as possible. But the precautions needed to furnish our cities with milk containing only a few thousand bacteria to the cubic centimeter are so expensive, that there seems to be no hope for this result until a radical change can come over the whole milk industry. This being the case, it is certainly desirable to be able to tell whether the bacteria present in such milk are those that render the milk suspicious or those that are perfectly harmless and, indeed, useful in their action. The number of bacteria may be reckoned by the hundreds of thousands, or even millions; but if the vast majority of them are the ordinary harmless lactic type, the milk may be, and probably is, perfectly wholesome in spite of the great quantities of bacteria present. On the other hand, the numbers may be much smaller; but if the percentage of lactic organisms is small, 10 or 15 per cent., and the total numbers are moderately high, the milk becomes immediately suspicious. It is not necessarily unwholesome even then, for most of these other organisms are harmless. But milk that contains great numbers, only a small percentage of which are lactic bacteria, is much more suspicious than milk with a large percentage of lactic bacteria, even though the latter sample may have a much greater total number of bacteria than the former.

From these facts, it will be apparent that the conclusions that are derived from the simple study of the numbers of bacteria in milk are not only incomplete, but frequently misleading. They would lead to the condemnation of many samples of perfectly wholesome milk, and they would pass as wholesome many a sample of milk that is decidedly unwholesome. All that can be determined from the numbers of bacteria in milk is a general knowledge of the age of the milk, the temperature at which it has been kept, and the cleanliness of the original conditions. Old milk, or milk kept too warm, or milk produced under uncleanly conditions, will contain large numbers of bacteria. But if we should find the numbers to be 20,000,000, we have no means of determining whether the milk is old, or has been kept too warm, or has been produced in uncleanly conditions. The conclusions which are to be drawn from the simple determination of numbers are so untrustworthy, that it is doubtful whether they are not at the present time worse than useless, since they may place emphasis upon points where emphasis should not be placed. If it were possible to carry on a bacteriologic analysis of milk which would differentiate species from one another, or even one which would differentiate the types of bacteria above referred to, we should then be able to obtain data of more decided value. Until a differential analysis is adopted, the interpretations that will be drawn from a bacteriologic study are bound to be misleading, and will be sure to result in conclusions which may be far from the facts. It is distinctly imperative, if our health boards are to do anything with the bacteriologic analysis of the milk of our city supply, that there should at least be adopted some method of distinguishing the normal lactic bacteria—which are sure to be present in the best of milk, and whose presence is not only harmless but frequently of distinct utility—from the other miscellaneous types of micro-organisms which render the milk suspicious, and to a considerable extent harmful.

THE CAUSATION, NATURE, AND PREVENTION OF THE SUMMER DIARRHEAS OF CHILDREN

BY ALFRED HAND, JR., M.D.
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THERE has been so much discussion of a proper nomenclature for the acute gastro-intestinal disorders of infants and so much dissatisfaction with all of the classifications proposed, that I have chosen in the title of this paper—or review, as it is mainly a compilation—that term which is the briefest and most comprehensive, and at the same time is as well understood as any other by the medical profession. I need not state the objections to the term "summer diarrhea," nor to classifications based on the supposed pathologic changes or on the anatomic distributions of the lesions. I may say that my own preference is for "enteritis" (with variations to gastro-enteritis and enterocolitis as the case may demand), for I believe that structural changes exist even in the mildest cases. The discussions as to nomenclature that have arisen may in a short time seem to be only of academic interest, and may remind us of those that were indulged in up to twenty years ago on the subjects of diphtheria and croup. As that field was cleared by researches in etiology, so it is not unreasonable to hope that we are now on the threshold of a similar result in the subject under discussion.

ETIOLOGY.—For a long time the summer diarrheas of infancy have been held to be infectious, and as one after another of the investigators—Booker, Baginsky, Escherich, Spiegelberg, and others—failed to identify a specific agent, the general belief has been that there were probably many different varieties of germs acting as exciting factors. The time, however, was not ripe, for the necessary preliminary discoveries had not been made, and no one had thought of connecting the tropical dysentery, generally believed to be due to the ameba, with a disease in the temperate zone apparently limited to infants. To establish this connection it is necessary to recall Shiga's discovery of a bacillus as the cause of acute dysentery (or of what is now known as the bacillary form) in Japan, and its confirmation by Flexner, in the Philippines. Stimulated by the latter, Duval and Bassett,[1] in the summer of 1902, isolated from the stools of 42 cases of summer diarrhea in infants a bacillus corresponding to Shiga's bacillus, and after careful study they believe the conclusion is justified "that the summer diarrheas of infants are caused by intestinal infection with Bacillus dysenteriæ Shiga, and therefore are etiologically identical with the acute bacillary dysentery of adults."

Duval and Bassett isolated the dysentery bacillus not only from the stools but also from scrapings of the intestinal mucosa at autopsy, and in one case from the mesenteric glands and the liver. In acute cases the bacilli were present usually in large numbers in the stools, but in the mild cases (fermental diarrheas?) and in those running a long course the detection of the bacilli was more difficult, owing to the antagonism of the normal intestinal bacteria. The dysentery bacillus isolated from these cases corresponded in morphology, cultural peculiarities, pathogenic properties, and reaction to specific serum, with the dysentery bacillus isolated by Shiga in Japan, Flexner in the Philippines, Kruse in Germany, and Vedder and Duval in the United States. Duval and Bassett state that "agglutinative reactions were obtained when the organisms were tested, (*a*) with the blood serum of the patients from whom they were secured, (*b*) with the serum of other infants suffering from summer diarrhea, (*c*) with the serum of adult patients with acute dysentery, and (*d*) with antidysenteric immune serum. The specific bacillus was not found in the stools of 25 healthy children, nor of those suffering with simple diarrhea, marasmus, and malnutrition; nor did the blood serum of these latter individuals agglutinate the dysenteric bacillus."

This publication aroused at once great interest not only in the medical profession but among the general public, especially as the report was made with the approval of Dr. William H. Welch. The publication came at a time when the disease was undergoing its usual autumnal retrogression, so that the general confirmation (or perhaps the rejection, which would certainly be unexpected) was necessarily deferred until the present summer, following which there will probably be a great increase in the literature on the subject. In the mean time an important contribution has been made by P. H. Hiss, Jr., and F. F. Russell[1] which deserves careful attention. They obtained from the colon, at two autopsies on cases of enterocolitis, cultures of a bacillus which bore a strong resemblance to the dysentery bacillus. They labelled this germ Bacillus "Y" and found that in form, absence of motility, staining properties, and growth on the ordinary media it behaved exactly as the dysentery bacillus, with the slight exception of forming a pellicle on broth—which is not the case with either the typhoid, the colon, or the dysentery bacillus. In studying the fermentative properties of these different germs toward various sugars, they found that Bacillus "Y" was like the dysentery bacillus in fermenting, with acid production, dextrose and galactose, and in being unable to ferment maltose, lactose, saccharose, and dextrin; as a point of differentiation, they found that like the typhoid bacillus it ferments mannite with acid production, which the dysentery bacillus does not; on the other hand, it may be distinguished from the typhoid bacillus by the latter's motility and power to ferment maltose and dextrin. The Bacillus "Y" therefore seems to be closely allied to the dysentery bacillus, but is also apparently related to the typhoid bacillus. A further study by Hiss of three cultures sent by Flexner, which had been isolated in Manila, showed that all of these, like the Bacillus "Y," fermented mannite, and two of them were even more closely related to the typhoid bacillus, like it fermenting maltose and dextrin, but being differentiated by lack of motility and by fermenting saccharose.

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[1] Amer. Med., September 13, 1902.

Hiss and Russell also found the Bacillus "Y" to have agglutinating reactions toward serum from human beings with dysentery, toward artificial dysentery serum (from a horse immunized to one of Flexner's cultures), and to artificial typhoid serum from a rabbit, the reactions in all cases being stronger and with higher dilutions than was the case with the cultures of Shiga's bacillus obtained from Kruse and Flexner. (It may be added here that none of Flexner's cultures agglutinate with natural typhoid serum.) The authors state in conclusion that "a description of this organism, apart from its connection, which may or may not be etiologic, with the case of diarrhea, has seemed to us important, since tests, too often relied upon, of its ability to agglutinate might lead to its being confounded with Shiga's bacillus. A subsequent use of such an organism for serum diagnosis might thus give rise not only to confusion in determining the 'specific' character of a disease, but to untoward results in the application of serum therapy. Its etiologic significance we have not been able to determine, but its close physiologic connection with the dysentery bacillus on one side and the bacillus of typhoid fever on the other make it not improbable that further research will show it to be associated etiologically with certain diarrheic or dysenteric attacks."

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[1] Med. News, February 14, 1903.

Whether the above differences between the dysentery bacillus and Bacillus "Y" are enough for basing the recognition of a distinct species is a question to be settled in the laboratory, the solution of which we may await with patience. Thus, with regard to the dysentery bacillus itself, differences exist between the cultures obtained by Shiga and those of Flexner and of Kruse, the majority of bacteriologists holding that these differences are minor and not to be considered as characteristic of distinct species. It is probable that the dysentery bacillus (like the colon bacillus) is rather a family with several forms or individuals which differ somewhat from one another, the main cultural peculiarities being the same, but the forms being distinguished by the above-mentioned fermentations and by the cross-agglutinations; that is, the serum of an animal immunized toward one form giving an agglutination with that form in higher dilution than with the others.

W. H. Park[1] arrives at similar conclusions after studying, with Carey, an epidemic of dysentery in a town (3000 population, 350 cases) with no sewage system and with badly contaminated well water. From a fatal case in an infant the true Shiga bacillus was isolated at the autopsy, and dysentery-like bacilli were found in the stools of several convalescents—the points of difference of the bacilli being in the formation of indol, fermentation of mannite, and agglutinating reactions. The authors believe that there may be more than one form of bacillus responsible for dysentery epidemics, and that if Shiga carries out the above culture and agglutination tests he will find that in Japan, as well as elsewhere, there are probably several varieties of Bacillus dysenteriæ.

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[1] Jour. of Med. Research, March, 1903.

L. W. Strong[1] also reports an epidemic of diarrhea at a seaside summer resort, where the sanitary conditions seemed perfect, the milk supply being the suspected source of infection; the bacillus isolated from four of the cases agreed with the Shiga form. Clinically there were several types of diarrhea—the author speaking of simple, non-bacterial diarrhea, fermental diarrhea, and ileocolitis, but he admits that no definite relation has been found between a special form of bacteria and any one variety of diarrhea, the same organism perhaps being able to cause a fermental diarrhea or an ileocolitis; the only way to determine the cause in various cases is by the serum reaction. The author refers to a possible origin for epidemics in the fact that the organism is viable in the soil even longer than the typhoid bacillus and so, by means of dust or pollution of water, a community may become infected from one case, if proper disinfection is not enforced.

The spread of the disease by infected dust has been referred to previously by F. J. Waldo,[2] who attributed the summer diarrheas in part to the germs present in horse-dung, the Bacillus coli communis, and the Bacillus enteritidis sporogenes, which are blown off the street surface by the wind and easily infect food.

H. D. Chapin[3] also has shown that in 1901 there were 6115 deaths in New York from summer diarrhea, or nearly twice as many as in 1900, which he attributes to the fact that in 1901 the streets of the borough of Manhattan were torn up very extensively, filling the air with dust. I do not know that the local conditions in New York were the same in that regard in the summer of 1902, which, as is generally known, was an unusually cool summer, and yet up to the middle of July[4] there had been 254 more deaths from summer diarrhea in infants than during the corresponding period in 1901. The difference in temperature for the two summers is shown by the death from heat-stroke, there being 1267 deaths in 1901, and only 31 up to the middle of July, 1902. Charts plotted by the Board of Health showing the temperature, humidity, and deaths for each day, bore no relation to one another warranting any conclusions. But as A. C. Cotton[5] pointed out in discussing Chapin's paper, deaths on one day may have been due to conditions entirely different on some preceding day.

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[1] Boston Med. and Surg. Jr., March 26, 1903.

[2] Lancet, May 26, 1900.

[3] Arch. of Pediat., July, 1902.

[4] Amer. Med., August 23, 1902, p. 284.

[5] Arch. of Pediat., July, 1902.

The general predisposing causes of the disease are given by A. Newsholme,[1] who draws his conclusions from an extensive study of statistics. He believes that the fundamental favoring condition is an unclean soil, from which the specific germ, not yet isolated, infects the air and is swallowed, usually with food, especially milk, and he therefore finds that communities without the water disposal of sewage or careful scavenger systems have a high mortality from diarrhea, as do those communities situated on a loose rather than a rocky soil; the disease affects chiefly children of the laboring classes in cities, the industrial occupations of the mothers having a possible influence; and there is a general direct relationship between temperature and diarrhea, and an inverse relationship between rainfall and diarrhea.

H. Heiman [2] refers, among other points, to a predisposing cause acting on the individual rather than on the community, comprising improper feeding (especially overfeeding), children as well as infants being unable to digest as much food during the summer as during the cooler seasons, the indication being, therefore, to reduce the solids of the diet and to increase the water.

The subject of diet leads to a consideration of milk as a factor in the production of summer diarrhea, and whatever other predisposing causes exist, every one is agreed that a rise in the mean temperature to 60° F., with the resulting increased bacterial development in milk, and an increase in the diarrheas of infants is more than a coincidence. Some points concerning the general milk supply of cities are well brought out by J. H. M. Knox and V. H. Bassett,[3] who examined over 54 samples of milk obtained in Baltimore. Their conclusions (which will doubtless hold for any city, although the gospel of clean milk is making encouraging headway in some places, and the laws relative to adulteration are not always dead letters) are that a large part of the milk supplied to the poor (of Baltimore) is unfit for use for infants, owing to adulteration with water, to the presence of chemical preservatives and coloring matters, to the high acid content, and to the great numbers of bacteria; they urge the education of the producers and dealers in measures of cleanliness, and the education of the people as to the need for clean milk in order to support the regulations of the Board of Health; and they believe that if proper milk could be furnished to the poor in summer there would be a decrease in the death-rate from summer diarrhea

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[1] The Practitioner, August, 1902.

[2] Arch. of Pediat., June, 1902.

[3] Maryland Med. Jour., June, 1902.

The influence of the rising temperature on the prevalence of summer diarrhea is well shown by the number of cases of diarrhea in my dispensary at the Children's Hospital during May, June, and July for the last seven years. In that time, out of a total of 2646 cases there were 635 cases of enterocolitis in children under 2½ years of age, or 24 per cent. In these months the morbidity was as follows:

Of 2646 cases, 28 per cent. came in May, 35 per cent. came in June, and 37 per cent. came in July. The diarrheas were distributed as follows: Of 635 diarrhea cases, 12 per cent. came in May, 36 per cent. came in June, and 52 per cent. came in July.

A closer analysis of these figures showed some interesting variations, without available data to explain them. Thus in May, 1901, 23 per cent. were diarrheas; in May, 1902, 2 per cent.; in May, 1900, the percentage, 12, was a little above the average for the 7 years, 9.7 per cent., and yet the succeeding June—20 per cent.—was the lowest but one of the seven Junes, no cases of diarrhea coming for the first 11 days. While collecting these data, I was struck with the way in which the cases came in groups of 4, 5, or 6, but there is nothing unusual in this, as the same fact holds good for practically all of the acute infections. A study of the weather conditions for the corresponding days would probably show nothing noteworthy, for the patients were rarely brought on the first day of the illness.

Pathology.—A. Baginsky,[1] in reviewing the studies of the last 10 years in the pathologic histology of gastro-enteritis, compares the completeness of the knowledge of this part with the unsatisfactory state of that of the etiology; and this completeness has been accomplished in spite of the difficulty of obtaining the tissues before cadaveric changes have taken place. These studies have corrected the former belief that, inasmuch as the macroscopic appearance of the intestinal walls was so often negative, histologic changes did not exist, the symptoms being the result of toxemia. Carefully prepared sections show, in various parts of the intestinal tract, lesions which can be compared in degree to those affecting other organs in other diseases. From a clinical stand-point, however, it is probable that in some cases the lesions do not advance beyond a hyperemia, such patients having signs of indigestion with vomiting and diarrhea, or constipation with colic and tympany, the attacks subsiding in a day or two with regulation of the diet. Nevertheless, such symptoms may be only the forerunners and accompaniments of profound lesions, which affect first that part of the intestinal wall nearest the lumen, because the harmful agents are in the vast majority of cases introduced into the alimentary canal through the mouth.

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[1] Arch. f. Kinderheilk., vol. xxxii.

The lesions that are found consist of two main types, one affecting the epithelial cells, the so-called catarrhal changes, the other affecting the lymphatic structures, the so-called follicular changes. The lesions usually occur in limited areas, especially in the ileum and colon, and are sometimes so fused that the original type is obscured.

The earliest catarrhal changes, which have been found in cases presenting only mild dyspeptic symptoms (death occurring from some other cause), consist in an infiltration of small round cells into the mucosa, with an outpouring of mucus, and cloudy swelling of the epithelial cells. The only difference in the lesions in ordinary dyspeptic diarrhea and in cholera infantum is in degree, the latter showing intense catarrhal changes in the cells, with loss of the epithelium in places. In the cases that run a longer course there is a proportionate tendency for the lymphatic structures to become enlarged, and after the overlying epithelium has been shed, to form deep, crater-like ulcers, the submucosa and muscular layer showing areas of round-cell infiltration. With proper staining, bacteria are also seen, even in the deep layers. In young infants especially these changes are apt to lead to a hyperplastic, followed by an atrophic, condition, with a state of malnutrition developing, complicated by lesions of various organs, particularly the lung and middle ear.

The follicular changes, which have just been described as secondary to the catarrhal, may set in as a primary process, follicular enteritis being a distinct pathologic entity, although a corresponding clinical type cannot be recognized. That the lymphatic structures may be affected before the epithelium is due to the thinness of the intestinal wall, the lymph-nodes here not acting in the way that other lymphatic glands elsewhere act, as sieves or barricades to germs that have gained entrance through another tissue.

Prevention.—Viewing summer diarrhea as an infectious disease, whether specific or due to several kinds of germs makes little difference, the prevention will naturally be along what may be called predisposing and direct lines.

To avoid predisposition of infants to the disease, two groups of measures are to be adopted. The one may be called personal, and will include the domestic care, such as should exist in every nursery, however humble, comprising cleanliness of nipples, bottles, etc. These are discussed in another paper in this issue and need not be detailed here. The other group of preventive measures may be called general, and are those which are to be adopted toward the community as a whole. These include general sanitation, with certain special measures, and care of the milk before it reaches the homes of the people. Under the head of general sanitation will be included, as having a direct bearing on the prevalence of summer diarrhea, the water supply, the disposal of sewage, the condition of the streets, and the existence of parks.

The Water Supply.—I need not dwell on the importance of pure water for children, for external as well as for internal use. As the majority of cases of summer diarrhea occur among that class of people who have not a superabundance of riches and are not able to indulge in the—in some places—luxurious necessity of house filters, it is the urgent duty of every municipality to see that its water supply is pure. It is not necessary to advance argument to sustain the assertion that epidemics of typhoid fever and dysentery are usually set up by polluted water. Park has shown that the Shiga bacillus was present in an epidemic of dysentery undoubtedly dependent on polluted water, the majority of fatal cases being among children, and it is safe to state that some cases of enterocolitis, the proportion of which will be settled in a short time, are dependent directly on polluted water or indirectly on milk contaminated by the water used for cleansing of the cans or for adulteration. If the water supply of a community is not above suspicion, then all the water used for an infant and for the cleansing of the bottles and nipples should be boiled, although the prolonged use of boiled water for drinking purposes is to be deprecated.

Disposal of Sewage.—Newsholme has pointed out the greater prevalence of summer diarrhea in communities without a good sewage system, and it is reasonable to suppose that a certain proportion of diarrheas depend on the breathing of dust-laden, germ-carrying air. These germs may come, in part, from a sewage-saturated soil, and surface drainage and cesspools (unless the latter are properly constructed) are to be condemned in severe terms. The greatest danger from such unsanitary condition is, however, that of rendering the water supply dangerous. Reference may be made at this point to the importance, enhanced since Duval's discovery, and mentioned by L. W. Strong, of carefully disinfecting the discharges from every case of summer diarrhea.

Condition of the Streets.—Whether the tearing up of the streets of New York and the tremendous increase of summer diarrhea referred to by Chapin bore any relation to each other cannot be stated positively, but there must have been something more than coincidence; in support of this is the low mortality (I regret that I have not the exact figures) from this disease when Waring made New York clean. As a matter of economics, the cost of clean streets is probably returned many times over in the lives saved. The practice of sprinkling the streets and of flushing the gutters from the fire-plugs may seem a small matter, but in the severe heated spells of summer it is certainly a benefit and not without influence in saving life.

The existence of parks and other measures designed to give city dwellers opportunities for breathing fresh air accomplish more than mere pleasure-giving. Clinically the effect of sea air in cases of enterocolitis and cholera infantum is so striking and well known as to need no further elaboration. Scientifically it has been demonstrated by W. T. Longcope[1] that the complement-content of the blood, upon which its bactericidal property practically depends, is much greater after a vacation than before. The depressing effect of an uninterrupted residence in the city comes with unusual force on infants and children, and therefore there cannot be too many breathing-spaces in the city nor too many opportunities for escape from the stale air of the city by means of day excursions on the water or on the country trolley roads which now fortunately intersect the whole country, or, best of all, through the organizations for giving children, infants, and their mothers, a week or two of life in the country or at the seashore.

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[1] University of Pennsylvania Med. Bull., November, 1902.

Inspection of the Milk Supply.—A strong argument for viewing milk as the carrier of the infection which produces summer diarrhea is the effect of sterilization of milk on the prevalence of the disease. In the large cities it is now possible to obtain a milk of high standard of purity and cleanliness, this milk rarely needing sterilization, but the advanced cost necessary for the production of such milk limits its use. The general milk supply, which, as Knox has shown, is far from what it ought to be, may be much improved without increasing its cost. As physicians are perhaps in a better position than any other class to use their influence in this direction, I have thought that it would be of value to mention the points, armed with a knowledge of which we can go to the average dairyman and with a modicum of tact show him how his milk may be made a safe food for infants.

H. W. Conn [1] has systematized this subject so well that I cannot do better than to give a brief abstract of his views, referring the reader who may desire a more complete statement to his extremely interesting little book. Under the heading of how to reduce the bacterial contamination in milk, Conn recommends grooming of the cows two or three times a day, especially around the flanks; whitewashing of the stable twice a year, the use of clean bedding, removal of manure several times a day, and keeping the stable as clean as possible; a separate milking room is of great advantage; manure should not be piled within several hundred feet of the stable on account of the increase of bacteria in the air and of the presence of flies; the employees should be cleanly in their persons, and hands, arms, face, and beard should be thoroughly washed before each milking; a clean suit of wash-goods to be put on for the milking is of great help; any employee coming in contact with a case of infectious disease ought to be temporarily absented from the dairy; the milk-vessels ought to be steamed, if possible; if the dairy has not this facility, the vessels should be scrubbed with soda water after each use, then rinsed with boiling water and turned upside down to drain; they should not be wiped with cloths nor rinsed with cold water after using the boiling water; the water supply for this purpose is of greater importance than that for the cattle to drink, and no well located near a house or a privy-vault is safe from contamination; the bacterial content of the milk can be greatly diminished if a suitable milk-pail is used, one of the best having a special cover with a small opening at one side over which a sterile piece of cheesecloth can be placed, thus straining the milk as it falls into the pail. Other points connected with the milking are to wipe the under side of the cow's body and the udder, but not the teats, with a wet cloth just before milking and to fasten the cow's tail so that it cannot be swung over the pail. Practices to be condemned are the use of dry feed such as hay just before or during milking, and " wet-milking" or having the milker's hands wet, this merely washing off germs from the hands and teats into the milk. The final steps in the care of the milk on the part of the dairyman are the placing in proper receptacles and the cooling, which should be immediate. If it is necessary to carry the milk any distance to the milk-room, the pail should be covered; the best receptacles are clean glass bottles.

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[1] Bacteria in Milk and its Products, Philadelphia, Blakiston, 1903.

As regards the direct prevention of summer diarrhea, at present a purely speculative question, it is safe to assert that some, at least, of the summer diarrheas are due to the Shiga bacillus. With cultures of this bacillus a curative serum may be obtained, but whether this will be feasible for preventive inoculations or not may depend on whether it has antitoxic or antibacterial properties; if the former, its value as a preventive will probably be greater, but perhaps even then it will not be adapted for prevention on a large scale any more than is the case with the diphtheria antitoxin, because of the transitory character of its immunity. For the good of civilization, it is to be hoped that the curative serum will not be of great use in preventing summer diarrhea, the true prevention being along the lines indicated above, and anything which would tend to make those general hygienic measures less necessary may be looked on as a public calamity.

THE SYMPTOMATOLOGY OF THE SUMMER DIARRHEAS OF CHILDREN

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INFANCY and childhood are the periods of life in which summer diarrheas are most prevalent and fatal. In children of all ages a transient form may occur at any season, with decided predilection, however, for the heated term. In midwinter occasional attacks of severe, although usually transient, diarrhea without fatal tendency are seen. It is in the summer months that enteric disorders, with diarrhea as a common symptom, are most fatal. This clinical fact is so well recognized, indeed, that the term, summer diarrhea, has gained a prominent place in the nosology of early life.

The high rate of mortality among infants and children during the summer months, resulting from disorders of which diarrhea is an accompaniment, exceeds that of all other diseases during the same period. Comparative statistics show that the greatest susceptibility to summer diarrhea is found between the ages of 6 and 18 months, during which time the death-rate is manifold that seen at any other age. The well-known mother's dread of the second summer is a clinical recognition of this fact.

The death-rate seems to divide this subject into two classes: (1) that of early infancy, and (2) that of late infancy. It is a common clinical observation that the persistency of digestive disturbance, due to the introduction of an irritant, diminishes with the age of the child after two years. So that with increasing age there is a steady diminution in the severity of diarrheal disorders and a corresponding decrease in mortality from this cause. The first class alluded to includes infants during the milk-feeding period up to about the middle of the second year, while the second class includes infants from 18 to 36 months of age. After the latter age severe attacks of diarrhea are so infrequent as rarely to require the attendance of a physician.

In these disorders the onset is sometimes sudden, being ushered in by a convulsion, vomiting, and diarrhea. There is usually high temperature, thirst, and nausea, which makes the infant snatch at food, but push it quickly aside as soon as a few mouthfuls have been swallowed. Restlessness is nearly always present. The stools may change suddenly, or there may be an interval of two or three days of preceding departure from the normal in frequency and character. There may be a great deal of colicky pain.

The infant while apparently in perfect health may develop vomiting, the matter ejected being at first the contents of the stomach but little changed—highly acid or in dense curds; this is soon followed, however, by a watery, mucoid, and, rarely, later, by bile-stained fluid—less acid or even alkaline in reaction. Diarrhea frequently accompanies and invariably follows the onset of the disturbance. The first dejections may be nearly normal, but they quickly change, showing evidence of fermentation and later of putrefactive processes. Finally they become serous. The odor of the stool varies from the normal acid to intensely fecal and later to a sickening musty smell.

The infant, apparently well nourished and playful, quickly shows signs of systemic disturbance in fretful, peevish irritability and restlessness, with head rocking, anxious facies, persistent whining cry, pyrexia, dry mouth, softened flabby tissues, and muscular atony. The depressed fontanel, sunken, lusterless eyes, sharply defined temporals, drawn features, with the intense thirst give evidence of the great drain of fluids. The abdomen, at first tympanitic, may soon become flabby and depressed and the extremities cold, although the rectal temperature may be from 103° to 106° F. Convulsions may usher in the attack, follow the initial vomiting by a few hours, appear only at the final stage, or occasionally they may not develop. Within 24 hours from the onset, and frequently earlier, the algid stage is reached, with pallor and cold superficies, though the internal temperature may be high. The restlessness changes to a sopor, accompanied by hyperesthesia and irritability when disturbed, but rapidly subsides into semi-coma with shallow breathing, collapsed veins, pulseless radials, half-closed, filmy eyes, and terminates in death by convulsions or asthenia.

This picture presents symptoms of acute poisoning from the gastro-enteric tract, occurring in a state of apparent perfect health and terminating fatally in from 24 to 48 hours. Though the well-nourished infant is not exempt from this form of acute poisoning, it occurs with so much greater frequency among those who show evidence of malnutrition that the latter condition may well be considered a predisposing cause to this form of intoxication. Fortunately cholera infantum as just presented, with its almost invariably fatal termination, is by no means common. It is extremely interesting to note the varying opinions of the most competent observers as to its frequency. One eminent observer of wide experience among diseases of children stated recently in a public address that he had seen but three cases of undoubted cholera infantum. Other writers of equal prominence speak of its occurrence as more common, and claim occasional success for certain therapeutic measures adopted in certain cases.

These widely different opinions as to the frequency and fatality of cholera infantum may be reconciled only upon the ground that we have no common standard for the diagnosis of this grave disorder. The prevailing opinion seems to be that so-called cholera infantum is extremely rare, and almost invariably fatal, and that against it no therapeutic procedure has so far proved efficient.

So far as clinical evidence goes, cholera infantum may be considered an intense form of gastro-enteritis, or a dyspeptic diarrhea plus some unknown intoxication. Clinically the line cannot be sharply drawn between some cases of dyspeptic diarrhea and cholera infantum. It is within every clinician's experience that occasional cases presenting all the symptoms of so-called cholera infantum, from the sudden onset to the serous diarrhea and vomiting, seem to yield to treatment and ultimately recover. Who shall say that these were not cases of cholera infantum? Is there not a tendency to restrict that diagnosis to fatal cases? Symptomatically the differentiation between the various form of "summer complaint" cannot be made. That bacteriology promises an early solution to some of these questions is believed by many, and the attention of the profession is turned with renewed interest to the special work of Booker, Flexner, Duval, Bassett, and others in their investigations along this line.

The commonest form of summer diarrhea is so evidently the result of acute indigestion that some writers have classified it separately as the dyspeptic variety. This classification seems justifiable when we recall the frequent history of the ingestion of substances known to be inimical to normal digestion. In many other cases which give no record of dietetic error we are led to conclude by analogy of onset, symptoms, and progress that perverted or disturbed digestion from causes not apparent may be responsible for a large proportion of summer diarrheas. It is the common experience of every physician to meet cases of sudden onset of vomiting and diarrhea, with temperature varying from slight elevation to a high degree of pyrexia, with or without history of improper feeding, in which relief with absolute recovery follows the exhibition of one or more doses of castor oil and the withdrawal of food for 24 hours.

The diagnosis of diarrhea from acute indigestion seems to meet all the pathologic and therapeutic requirements. Unfortunately, however, in many cases the simple treatment outlined above is not applied, or if applied fails to secure the desired results, and the case develops a more obstinate form of gastro-intestinal disturbance. There is continuation of pyrexia, with colicky pain, more or less flatulence, and frequent dejections of foul, acid, greenish stools of varying inconsistency. The vomiting may cease, but the diarrhea persists with increasing tenesmus, and the stools become mucoid, bloody or serous, and either fetid or odorless. From the common onset the resultant disorder may be named according to the area apparently most involved as gastro-enteritis, enterocolitis, ileocolitis, or colitis with or without proctitis. It is amply in evidence that obstinate enteritis with or without anatomic change in the mucosa of the gut, or even membranous or ulcerative colitis, may be the last expression of an acute attack of indigestion in an infant.

The systemic disturbance which seems to be an expression of intoxication from the stomach and bowels varies widely as the diarrheal disorder locates with greater or less intensity upon this or that portion of the digestive tract. The early cessation of vomiting is frequently accompanied by the subsidence of pyrexia, with improvement in the general muscular tone. Apparently systemic disturbance diminishes proportionately with the distance from the stomach at which the burden of the disorder seems to fall. Thus, in most obstinate cases of colitis with extensive lesions of the mucosa, nutrition is frequently maintained to a remarkable degree, the child showing but little evidence of systemic intoxication; whereas a much less obstinate disorder of the small intestine may cause prostration, with symptoms of general infection. In other words, the more active the digestive area involved the more profound the constitutional disturbance and the greater the tendency to prostration and collapse.

Of the complications of summer diarrhea, the most common in young infants is stomatitis from simple catarrhal to the obstinate ulcerative variety. It becomes serious as it interferes with the ingestion of food and increases the restlessness and discomfort, and its persistency adds to the gravity of the prognosis. This complication is most often seen when the gastric disturbance is marked, and its improvement is usually in direct ratio to the improvement in stomach digestion.

The cough and rapid respiration, so frequently seen in acute or subacute gastro-enteric disorders, is usually indicative of bronchopneumonia, which may or may not yield physical signs dependent upon the extent of lung involved. That it selects the most dependent portion of the lung adds to this infection the danger of mechanical hypostasis.

The quite frequent occurrence of albuminuria in patients with summer diarrhea has led observers to attribute the convulsions to uremia and the occasional so-called idiopathic edemas to nephritis. These conclusions, however, do not appear to be confirmed by the post-mortem findings. Functional cardiovascular conditions with the anemia rapidly induced by the colliquative discharges appear a rational explanation for many of the cerebral phenomena and the edemas of dependent parts.

Lesions of the integument, from mere dryness or ordinary intertrigo to the various forms of urticaria, furunculosis, and local necrosis, are met with most frequently in the subacute and chronic diarrheal disorders. Whatever the local treatment applied, they yield only to the improved nutrition of convalescence.

A prolonged attack of summer complaint rarely leaves the child free from evidences of retarded development, which in many instances is never fully compensated. The picture of a two-year-old set back to the plane of a yearling, both as to digestive and muscular functions, is a familiar one, and the attempt to adapt his hygiene to age instead of to condition is a common error.

Perhaps the most important effect of severe summer complaint upon the survivors is the feeble resistance to infection from lowered vitality, so that the danger from intercurrent diseases is manifold. The special hygiene for these little convalescents is a subject too frequently dismissed with the doctor at the conclusion of the enteric disorder.

It is safe to say that the fatalities of summer diarrheas are seen very rarely among the exclusively breast-fed infants. They may starve to death from insufficient or impoverished breast milk or die from inherited dyscrasias; they may show some of the symptoms of chronic indigestion; they may exhibit symptoms of acute or subacute digestive disturbance as a result of palpable errors in hygiene; or they may be destroyed by some overwhelming intoxication, presumably due to sudden changes in the constitution of the breast milk resultant from physical or mental disturbance in the nurse; but in none of these instances is the symptom diarrhea essentially a summer diarrhea. Moreover, it is a common observation that the diarrheas of the exclusively breast-fed are usually tractable, regardless of season, in otherwise healthy infants by the application of the well-known principles of hygiene.

The startling mortality from summer diarrheas is confined almost entirely to bottle-fed babies. These facts are so generally established that when pronounced diarrheal symptoms appear in an infant at the breast the first interrogations are directed toward the possibility of the ingestion of material other than breast milk. A careful study of the infant's hygiene, with examination of the dejecta after colonic flushings, frequently disproves the statement that the child's diet included nothing but breast milk. Statistics show that in the fatal cases of summer diarrheas less than 3 per cent. were exclusively breast fed. I know of no clinical phase of this subject that begins to rival in importance this one fact—that summer diarrhea, with rare exceptions, means practically summer artificial feeding.

In view of our knowledge of summer diarrhea, the question of feeding, then, is of paramount importance. A neglect to use every means to secure for the infant natural food cannot be atoned for by the most energetic therapy yet devised. Many of the obstacles to natural feeding are insuperable, or even formidable, only as long as the lay mind is asleep to the fact that her physician knows how to prevent fatal summer diarrheas in the infant. When the responsibility for the consignment of the infant to a method of feeding that diminishes his chances of living twofold more than inoculation with smallpox virus, or threefold more than the infection of measles, is fully brought home to the physician the difficulties of breast nursing will become proportionately insignificant. The fact that some governments and municipalities now require, on an infant's death certificate, a statement as to the method of feeding shows an awakening to the importance of this matter. How long before the same line of inquiry shall require an explanation as to why the infant was subjected to the manifold dangers of artificial feeding?

I would like to call attention to the fact that no united or concentrated effort has been made to cope with this, whereas an immense amount of time and work has been given to the subject of artificial feeding, with results that only emphasize our inability to rival nature's method. The paramount importance of breast feeding has been dismissed with here and there only complimentary mention. To be sure, isolated instances in infants' asylums and foundlings' homes appear in which relief was sought and found in wet nursing with a prompt decrease in the appalling death-rate in proportion to the breast milk supplied. But thus far no organized effort through any professional or lay body has yet, to my knowledge, been made to secure to the infant his right to nourishment from the breast.

Thousands of breasts are being dried up artificially or by neglect of function while thousands of babies succumb to the disorders for which this wasted product is the only known prophylactic. The treatment of summer complaint is so essentially dietetic that the few drugs indicated may be regarded as merely supplemental.

THE DIETETIC TREATMENT OF THE SUMMER DIARRHEAS OF CHILDREN

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The dietetic treatment of the summer diarrheas of children naturally falls into two periods: (1) That of the active stage of the disease; and (2) that of the period of convalescence and subsequent return to health. To these logically should be added the treatment during the period preceding the attack, for it is at this time that a judicious control of the diet and a supervision of all the details of feeding may achieve what is after all the most brilliant therapeutic success, namely, prevention. Many a child suffers from summer diarrhea because of the mother's ignorance of some of the simplest rules of artificial feeding; and in many instances the burden of responsibility rests with the physician who has neglected to warn her of the dangers of summer weather and to instruct her in the means of best avoiding them. Prophylaxis, therefore, rightly constitutes an extremely important division of the subject, and will first claim attention.

PROPHYLACTIC TREATMENT

The cardinal principle of prophylaxis lies in the use of pure milk as the basis of the infant's food. By this is meant the mixed milk of a herd of perfectly healthy tuberculin-tested cows, which contains a fairly constant average percentage of fat, proteids, and sugar; which is free from adulteration with artificial coloring, preservatives, or diluting water; and which contains a minimum count of bacteria of all varieties, with entire absence of any variety of accidental pathogenic organism. As a means to this end the physician should insist upon rigid periodical inspection, by a competent veterinarian, of the health of the cows, and of the sanitary conditions of the dairy buildings and pasture; upon the personal health of the dairy attendants and their freedom from contamination by contagious diseases; and upon the most scrupulous cleanliness and asepsis in every step of the preparation of the milk for delivery, which includes sterilization of all implements and vessels coming in contact with the milk, and the plentiful use of ice for refrigeration from the time the milk is bottled at the dairy to the moment of its delivery to the consumer.

It is needless to say that much of the milk used for infant feeding in summer weather fails to meet the requirements of these modern methods of dairy management; and that in this feature alone an inestimable safeguard is thrown around the infant whose food supply comes from such a carefully protected source. Such milk therefore requires special facilities and appliances which add considerably to the expense of production, so that it costs the consumer approximately twice as much as milk from the ordinary dairy. This, however, is of little moment in comparison with the heavy expenses and the dangers of a severe illness; and the writer has usually found that even in dispensary practice mothers are only too glad to get the best certified milk, rather than run increased risk with the grade of milk they have previously been using.

In country practice it usually happens that milk meeting all these requirements cannot be obtained, and too often the physician is not keenly alive to their importance in the success of his own efforts in feeding infant patients. It will then be necessary to make arrangements with the most progressive dairyman in the immediate neighborhood to deliver the milk with as little delay as possible after the milking; and, if it can be arranged, to have two deliveries daily, immediately after the morning and evening milkings. Under these conditions it will be best for the mother to provide two sets of cans or, better, bottles, for her own use, assuming herself the important duty of cleansing and sterilizing, and delivering the clean vessels to the milkman at the time of his visits. If bottles are used, clean pasteboard disks, which may be obtained from dealers in dairy supplies, should be fitted to the mouth of the bottles to protect them from dust and accidental contamination during transit, fresh disks in a sealed wrapper being given to the dairyman to seal the bottles after the milk has been put into them. As a check on carelessness, these disks may be marked differently, so that the substitution of once-used disks may be readily detected. In this way the milk given to the baby will be fresh, and even that in the last bottle used before the new supply is delivered need never be more than 12 to 15 hours old. Freshness under these conditions is the all-important consideration, and even "one-cow's" milk coming from the immediate neighborhood will usually be preferable to the milk of a herd which must be brought several miles through the hot atmosphere without the use of ice. Refrigeration with ice, however, is such a necessary prerequisite for the preservation of milk in hot weather that a strong effort should be made to have the dairyman supply it, even if at an increased cost.

In the city it is highly important to insist that the milk should be delivered directly into the hands of a responsible member of the household, so that it may be put upon ice at once, for it is frequently the practice of milkmen to leave the jar of milk on the doorstep very early in the morning, often several hours before any of the household is awake. If this arrangement cannot be made, a box of ice should be provided, in which the milk jars may be placed and kept cold until convenient to transfer them to the refrigerator. Unless these precautions be observed, bacteria will rapidly multiply as the temperature of the milk rises during this exposure to the heated atmosphere, and even the purest grade of milk may become unsafe for infant feeding before it is prepared for use. Consideration of this fact also prompts the obvious suggestion that after delivery not an instant's delay should occur in transferring the milk jars to the refrigerator. Carelessness of servants in this matter should not be tolerated.

What has been said of the purity and freshness of milk applies with even greater force to cream. As ordinarily sold, gravity (skimmed) cream is unsafe for infant-feeding at any season of the year because of its age and its exposure to the air during the setting, but in hot weather its use is extremely dangerous. In many cases of milk poisoning it is the cream and not the fresher milk which is to blame. These objections do not hold against centrifugal cream from a pure milk, and when possible such cream from a first-class dairy should be secured. Lacking this, it will be safest to make use of the cream layer of fresh bottled milk, by skimming or siphoning, or by obtaining cream of the desired fat percentage by means of the dipper device suggested by Dr. Henry Dwight Chapin, of New York.

During extremely hot weather pasteurization or even sterilization of the food may be resorted to as an additional means of preservation; but it must not be forgotten that neither of these processes can make wholesome a milk already unfit for food, though it does retard further deleterious changes. For this reason commercial pasteurization or sterilization of milk is not a guarantee of safety from the ordinary dangers of milk feeding, and its exploitation by dealers suggests a lack of confidence in the original purity of their product.

An often unrecognized cause of deterioration of milk in the household may be traced to the rather common practice of heating the night bottle quite hot just before the parents retire, and keeping it warm in a cozy or in the bed itself until such time as the baby wakes for its feeding. No more favorable conditions for producing rapid souring of milk could be devised; and in a previous contribution to the subject[1] the writer has shown by experiment the startling rapidity with which bacteria may increase in a milk mixture kept for several hours under these favorable conditions.

Another precaution that may be helpful in very hot weather is to give no milk that has been kept over night from the supply of the day before, which at the time of delivery is likely to be already from 12 to 18 hours old. If the fresh supply of milk cannot be obtained before the first morning bottle is due, a bottle of sweetened albumin water or of condensed-milk mixture may be given for this feeding. In this way the child may escape even a mild milk infection, a result which is especially desirable when we remember how difficult it often is to overcome an infected condition of the intestine as long as the hot weather lasts.

Again, following the common hygienic law, it is usually desirable to decrease the strength of a baby's food mixture during the hot weather. A child who has been taking a milk mixture which is just within his digestive capacity may avoid a digestive diarrhea if the percentages of his mixture are somewhat reduced as hot weather begins. The simplest method by which this can be accomplished is by fractional dilution of his ordinary formula. If, for instance, he is taking a mixture of 6 ounces (180 c.c.) of any formula containing more than 1.5 per cent. of proteids, a half ounce to an ounce (15 to 30 c.c.) of this quantity may be poured out, and its bulk made good by the addition of an equal amount of diluent. By this plan it becomes a very easy matter to return to the original formula, when so desired, by gradually omitting the added diluent and increasing by equal amount the original mixture. In this way frequent recalculation of formulæ is avoided and the matter may be left to the judgment of the mother without requiring frequent supervision by the physician.

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[1] INTERNATIONAL CLINICS, Tenth Series, vol. iii, October, 1900.

In many instances the danger of serious milk poisoning may be avoided if, at the slightest sign of disturbance of the stomach or bowels, the mother previously has been instructed to stop milk feeding at once and to give a good dose of castor oil. This should be followed in the cases showing a tendency to greenness and curdiness of the stools by small doses of calomel, one-twentieth to one-tenth of a grain (0.003 to 0.006 gram) three or four times a day for two or three days. If the mother has been taught that her baby can live comfortably and safely on a diet of little more than water for several days, there will be no difficulty in persuading her to stop milk feeding for a time whenever the character of the stools shows change.

It seems scarcely necessary to refer to the popular fallacy that looseness of the bowels in summer is a necessary accompaniment of teething. As to the question of teething, however, a word might here be said. The nervous erethism attending the eruption of teeth may at times and in more susceptible infants produce a slight increase in the frequency of movements, but without other change in color or appearance than would be expected from the greater rapidity with which the food has passed through the intestinal canal. The diagnosis in every case, however, must be made by exclusion, and it is always safest to regard any case of irregularity of the bowels in hot weather as a beginning enterocolitis, and to treat it accordingly. In the presence of a summer diarrhea dentition becomes a factor of considerable importance, adding greatly to the child's discomfort and doubtless aggravating the nervous irritability of the bowel. Under these conditions, lancing of the gum is a rational procedure and is free from danger when performed with a sterilized knife, the little wound being bathed frequently with an antiseptic lotion for 48 hours following the operation.

Since the infant at the breast may suffer from an infectious enterocolitis, it is important to impress upon the mother the necessity for great care in cleansing the nipples before and after nursing, and in keeping the baby's mouth clean by frequent, but gentle, applications of a bland lotion, such as boric acid solution. The disgusting habit of moistening the nipple with saliva before putting the child to the breast should scarcely need mention, were it not so frequently noticed among mothers of foreign nationalities. The introduction of an unclean finger into a baby's mouth is often thoughtlessly encouraged even by physicians. I have seen an eminent physician do this during his visit to a hospital ward, after he had been examining children suffering from various infectious conditions.

It is presumed that the ordinary rules for cleansing nursing-bottles and every article that may contaminate the milk are too well known to need reiteration in this place.

DIETETIC TREATMENT DURING THE ATTACK

In the presence of a summer diarrhea, however mild the symptoms may be, the first dietetic principle is absolutely to interdict the use of cow's milk. This injunction must be strongly emphasized, lest the mother, in her anxiety, be tempted to give the child "a taste of milk" to prevent it from starving under this seemingly heroic treatment, or because the child objects to the change in taste and refuses to take the full amount of the substitute food. The reasons for the withdrawal of milk should be explained to her, and assurance given that there is no danger of starvation under the new plan of feeding. By enlisting her co-operation upon rational grounds much harm to the child may be prevented and meddlesome interference, without the physician's knowledge, may be avoided. In such cases, it should be explained, the continuance of milk feeding becomes a serious menace to the child by furnishing a constant fresh supply of the food which offers the most favorable culture medium for the bacteria already swarming in the intestinal canal. Milk starvation of the infant really means starvation of the milk bacteria, and as this culture medium ceases to be introduced into the infected bowel, the great bulk of these organisms perish for want of favorable cultural conditions. The next question is, What substitute for milk should be used, and how long should milk be withheld?

The general experience of physicians has proved that under these conditions cereal decoctions offer the most satisfactory substitute food, supplying at the same time a liberal quantity of water, which is needed to make good the loss of fluid from the bowel and to carry off noxious toxins through the kidneys. The quantity of fluid to be given in the 24 hours should approach that to which the child is accustomed in its daily food ration, and the intervals of feeding at first should be about two hours. Barley water is the decoction most generally useful, but rice water, acacia water, or a natural alkaline water like Vals or Vichy may be preferred. Cool sweetened egg-water may be most useful for a time, when the stomach is very irritable, but is apt to undergo putrefactive change in the bowel, if not well digested. It should be discontinued as soon as the stomach has become quiet. Barley water for this purpose should be made somewhat stronger than when used simply as a diluent. One or even two tablespoonfuls of washed pearl barley and a little salt may be added to a pint of water and boiled for half an hour, hot water added to make up the loss in boiling, and the mixture strained to remove the shells and other solid particles. Very few children, however, will readily take this insipid fluid, and it is necessary to add to it some flavoring substance. For the younger babies nothing has proved so satisfactory as a teaspoonful (5 c.c.) of panopepton or liquid peptonoids added to the barley water, the quantity of which should correspond with the usual meal. If the child objects to the taste of these concentrated foods, a teaspoonful (5 c.c.) of a liquid beef extract may be substituted.

For older children the barley water should be two or three times stronger and may be dextrinized, and for variety, chicken, beef, or mutton broth may be given occasionally, or a mixture of 1 or 2 ounces (30 to 60 c.c.) of broth and 4 or 5 ounces (120 to 150 c.c.) of the stronger barley water may be acceptable. Broths, however, are rather laxative in diarrheal conditions, and should be used sparingly. If the stomach is very intolerant of reasonable quantities of fluid, a preliminary lavage with normal salt solution should be made, and teaspoonful (5 c.c.) doses of raw beef juice used for a short time until the barley water can be taken. Even if an insufficient amount of fluid for a time can be taken by the mouth, the use of intestinal irrigation will go far toward supplying the deficiency of water in the tissues, for under these conditions a considerable amount of the irrigating fluid is absorbed from the bowel and reaches the blood, where it acts as a stimulant to the circulation, and especially to the kidneys.

The length of time during which fresh milk should be withheld will vary according to the degree of severity of the case and the promptness with which the discharges become normal in frequency and consistency. The most common mistake is to resume milk feeding too early, thereby furnishing material for reinfection within the intestinal canal, even though the milk itself may be perfectly sound. With babies over two months of age who are in good condition at the outset of an attack of summer diarrhea, fresh milk should be entirely withheld for from five or six days to a week, and in the severer cases considerably longer. Under the plan of feeding above suggested, a well-nourished healthy baby will show little evidence of serious starvation even at the end of a week. By this time, however, and in the milder cases much earlier, the bowel movements will have decidedly decreased in frequency, and other evidences of intestinal irritation will have subsided. Even before this time the baby may show signs of hunger which cannot be appeased by the watery diet, and it will become necessary to add something to the mixture to give it more body.

It is a fact of frequent observation in dispensary practice that babies with diarrhea who had been previously fed upon condensed milk could be put back upon this diet earlier and with greater safety than the babies fed upon fresh milk. This is doubtless to be attributed to the fact that condensed milk ordinarily is a sterile preparation, and that the percentages of fat and proteids in weak dilutions are quite low, both of which qualities for the time are of considerable importance.

It has therefore seemed of advantage to make use of this property of condensed milk by employing it in very weak dilutions for a time in preference to an immediate return to fresh milk. A dilution of 1 dram (4 c.c.) to 4 ounces (120 c.c.) of the barley water and concentrated food offers a satisfactory strength. In some instances even this weak mixture is not well digested, and then partial peptonization may be resorted to. At this time, too, certain of the proprietary foods may be used alternately with the condensed-milk mixture, unless increased frequency of movements follows their use. A very satisfactory substitute for these preparations may be made after an old German recipe, which the writer has used with considerable success: With one teaspoonful of best butter mix two teaspoonfuls of wheat flour, and brown thoroughly in a chafing-dish; then add half a pint of hot water and bring to a boil, stirring until the mixture becomes smooth, and flavor with salt. To this may be added a teaspoonful of a liquid beef extract; or beef tea or chicken broth may be used in place of the plain water. Serve warm. For older children an egg may be broken into the soup while quite hot and quickly stirred so as thoroughly to break it up and distribute it in fine flakes. Many children will take this soup with relish; it may be given in appropriate quantities, at different meals, twice a day.

An exception to the general rule of prolonged starvation must sometimes be made in the case of artificially fed infants under two or three months of age. As a rule, these babies when first presented for treatment are found to be weak and badly nourished from improper feeding, and therefore even a moderate starvation for more than 24 hours may cause a serious loss of vitality. In several instances coming under my observation in dispensary practice, dangerous collapse at the end of 24 hours has followed the withdrawal of milk food. In such cases it becomes a matter of necessity to fortify the infant's strength by cardiac and systemic stimulants, and after the initial cleansing of the gastro-intestinal canal to begin feeding with a sterile milk food, as a choice of the lesser evil. In these cases a weak condensed-milk mixture (1 in 32) or a diluted pasteurized whey made from a sound milk may be absolutely required at a very early stage of the disease.

In older children the dietetic treatment of a summer diarrhea presents less difficulty owing to the greater variety of foods that can be taken; but withdrawal of milk for a time is always imperative.

DIETETIC TREATMENT OF THE CONVALESCENCE

It must be remembered that after any but the mildest attack of enterocolitis the mucous membrane of the lower intestinal tract, and frequently that of the stomach and of the upper intestine as well, is in a state of subacute catarrh, and that its restoration to normal functional activity depends upon two conditions: (1) the constitutional peculiarities of the baby; and (2) adaptability of the food.

In many cases serious anatomic alterations in the secretory and absorptive structures of the intestine have taken place. Rhachitic infants and those of strumous or gouty tendencies are notably slow in recovering from the effects of an intestinal infection, while normal infants show much more rapid recuperative powers. The digestive power of the convalescent will therefore be conditioned both by his constitutional defects and by the extent of damage sustained by his digestive mucous membranes. For these reasons the treatment of convalescence often presents dietetic problems that far surpass in difficulty those of the acute stage of the disease. An infant that had previously been thriving on a simple modification of cow's milk may be totally unable to resume this diet, and in many instances it may be months before fresh cow's milk in any sufficiently nourishing proportions can be given. This fact, however, can be arrived at in any particular case only by cautious experiment.

If the acute attack has been skilfully and promptly handled, there is good reason to expect that a cautious return may be made to the usual diet very soon after the evidences of intestinal infection have disappeared. After the movements have become natural in consistency and frequency, the most satisfactory plan for returning to fresh milk diet is to prepare and pasteurize the milk mixture which the baby has previously taken, at first in quarter or half the usual amount for the day's feeding; and to begin by taking out not more than half an ounce (15 c.c.) from the bottle of condensed-milk mixture or other temporary food and replacing it by an equal quantity of the fresh-milk mixture. If this change is acceptable to the child's digestion, an ounce (30 c.c.) of the fresh-milk mixture may be substituted for an equal quantity of the temporary mixture, and so on, this gradual substitution being continued with varying rapidity until half of the feeding consists of the fresh-milk modification. After this the condensed milk or other food may be omitted altogether, and the feeding will now consist of a one-in-two dilution of the original mixture, which will contain just half of the original percentages. From this point the proportion of the milk mixture may be gradually increased, according to the digestion as shown by the bowel movements, until finally all the added diluent is omitted and the baby has returned to his usual bottle mixture.

This gradual substitution in many instances may be completed in a few days; in others, it may take from several weeks to a month; but in the class of difficult cases already referred to it will be found that even this rate of progress is too rapid. If partial peptonization of the mixture does not offer a remedy at this stage, it will be necessary to make a complete change in the plan of feeding.

In most of these cases the difficulty lies in the infant's inability to digest any but the lowest percentages of casein, and therefore resort must be made to a whey-and-cream modification, in which the percentage of caseinogen can be reduced to a very low figure, while satisfactory percentages of fat, lactalbumin, and sugar can be used. Almost any infant can digest pure whey or whey partly diluted with water. Whey made from whole milk and drained by gravity without squeezing contains approximately 1 per cent of fat. With this as a basis a rich cream may be cautiously added in increasing proportions until a fair percentage of fat is obtained; the richer the cream the lower the caseinogen percentage that will be supplied with any desired percentage of fat. For instance, a mixture of one ounce of a 32 per cent. cream and three ounces of water may be peptonized at 105° F. for from 10 to 15 minutes with one teaspoonful of peptogenic powder, and then raised quickly to 150° F. to check the process of peptonization and destroy further activity of the pancreatic ferment. A fluid dram of this diluted cream (representing one-fourth part of cream) may be added to sufficient whey, pure or diluted, to make up the quantity of the feeding, and enough sugar of milk to supply an appropriate percentage of sugar. If this agrees well, the quantity of cream mixture may be gradually increased by dram additions until the cream mixture constitutes one-fourth and the whey three-fourths of the mixture (in which there is approximately 2.75 per cent. fat, 0.79 per cent. lactalbumin, and 0.16 per cent. caseinogen). Such a mixture contains quite a fair amount of nourishment and is very readily digestible. At this point of progress further additions of cream may be stopped and small quantities of whole milk added, equal quantities of whey being left out. In this manner the whey may be gradually replaced by milk until the mixture becomes a simple cream, milk, and water mixture. When the whey has been reduced to one-quarter of the total mixture, a weaker cream should be used (about 20 per cent.). Peptonization of the cream mixture may be stopped after the addition of milk begins, or the process may be continued for a time, the necessary quantity of milk being added to the cream dilution before the peptonization. When unpeptonized cream or milk is added to whey, it is important to remember that the whey should be previously raised to 150° F. to destroy the excess of rennin, otherwise the milk and cream will be precipitated as curds in the mixture. When the cream or cream and milk have been previously peptonized, this preliminary heating of the whey is not necessary, but should be carried out for its pasteurizing effect.

Finally, in a small proportion of these difficult cases it will be found that no fresh-milk food other than whey can be tolerated, although the infant may have thriven on an ordinary milk-and-cream mixture before the attack of diarrhea. Under these circumstances we may be obliged for a time to continue condensed milk, to which small additions of fresh cream after a time may be made. In some few cases, indeed, a weak condensed milk with one of the proprietary foods containing dried milk may be the only form of food offering satisfactory nourishment at this stage of convalescence, and we may be obliged to wait patiently until the child shows himself again able to digest fresh milk, added at first in small quantities to the other mixture.

In conclusion, the writer cannot too emphatically commend the excellent results to be obtained during this stage of convalescence from the severer grades of the disease in young infants by a temporary resort to partial or complete wet-nursing. Provided the woman be in good health and her baby thriving, no theoretical objections as to differences between the age of the sick baby and the period of the nurse's lactation need be taken into account, provided, of course, the colostrum period has been passed. Even if the woman cannot supply sufficient milk for the whole nourishment of the little patient, one, two, or three nursings a day will be of inestimable aid; and even if she can spare only a little milk, say 5 to 8 ounces a day, this may be used with a diluent in a sort of human milk modification, the amount available being apportioned as equally as possible in the bottles of the artificial mixture. A few ounces of human milk, with the balance of the bottle made up of pasteurized whey, will in the more serious cases supply a form of nourishment for a limited period that may save a life. In these cases the human milk acts not only as a food, but also as a powerful vital tonic and hematinic.

THE TREATMENT OF THE SUMMER DIARRHEAS OF CHILDREN

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Within the last few years notable advances have been made in our knowledge of the prevention and proper care of the various pathologic conditions classed, for the want of a more exact term, under the name of summer diarrhea. The results, while lacking the brilliancy that surrounds the treatment of a disease by a specific remedy, are nevertheless clearly shown (in the vital statistics of large cities) by a steadily decreasing death-rate from these diseases. Furthermore, the wide dissemination and general acceptance by the medical profession of certain fundamental principles of prophylaxis and treatment have served to render the prognosis much more favorable, even in the most severe and seemingly hopeless cases.

While the prevention of summer diarrhea is based almost wholly on the results of bacteriologic investigations, especially of the food and the milk supply, the treatment of actual cases as carried on to-day is founded on neither pathologic nor bacteriologic reports, but is the result of patient observation and experiment conducted over many years by the clinician, and is very largely symptomatic.

Clinically, what are we to include under the term summer diarrhea? However unscientific may be the proposed classification, until we have a more exact knowledge of the underlying causes of the different varieties of these diarrheas, they may be advantageously divided into: (1) Cases of intestinal indigestion, (2) cases of intestinal infection, and (3) the different varieties of enterocolitis. Indeed, a fair proportion of the cases met with will come under all three of these heads—in which event the case may justly be regarded clinically as three stages of a single disease.

The more one sees of the pathologic conditions of the gastroenteric tract, in fatal cases, the more he becomes impressed with the wide discrepancy between the clinical symptoms and the post-mortem changes. The most overwhelming attacks often show very few grave pathologic changes, while, on the contrary, those giving but mild enteric symptoms often show very marked lesions. Such discrepancies may be explained, no doubt, by the great intensity of certain infections which cause death by toxic absorption before changes can occur, while on the other hand it is possible to have a marked catarrhal or croupous condition with only mild toxemia, which kills slowly through the formation of chronic changes in the enteric tract. For these reasons in many cases it is not safe to feel confident of the exact condition of the intestine, and of necessity one is compelled to treat the disease not as he imagines the lesions, but the various symptoms as they arise.

It is in the large cities that the scourge of summer diarrhea is most felt, and it is to them that we must look for means of controlling it. Since it is not reasonable to suppose that the type of the disease has become less virulent, how are we to account for the steadily decreasing death-rate from this cause?

Dr. Louis C. Ager,[1] in a recent paper on the infantile mortality in Brooklyn, to which I shall have frequent occasion to refer, shows from a study of the health records that the death-rate in New York city from diarrheal diseases within the past 20 years has been more than cut in half, and also that the death-rate in the borough of Manhattan is much less than that of Brooklyn, Richmond, and the Bronx, in spite of the less concentrated population of the last three mentioned.

The following table shows the number of deaths from diarrheal diseases in children under 5 years of age in 100,000 of total population (Ager):

	Brooklyn.	Manhattan.
1898	224	191
1899	180	142
1900	196	160
1901	194	161

Thus there is a difference of from 30 to 40 in each 100,000 inhabitants in favor of New York. Ager accounts for this in brief by the better sanitary regulations, the better paved and cleaned streets, and the greater knowledge of the people in the feeding and care of their children in the borough of Manhattan.

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[1] Brooklyn Med. Jour., February, 1903.

Notwithstanding our lack of exact knowledge concerning the bacteriologic cause of these conditions, no one can doubt that the past year has brought us nearer the truth, that is, that a good proportion of the severe cases of enterocolitis are caused by a specific germ, or more probably group of germs, and if we accept this theory it will not be difficult to account for the improvement in the death-rate just noted.

At present we know little of these germs within the body, and practically nothing of them outside of it, and yet we have strong reasons to suppose that they thrive under filthy conditions of all kinds; that soil soaked with decaying animal and vegetable matter is favorable to their development, especially during the heat of summer.

Ballard, of London, has pointed out that when the ground temperature (that is, the temperature taken 4 feet below the earth's surface) reaches 56° F., cases of summer diarrhea begin to appear, and that they increase and decrease in number with the rise and fall of this ground temperature.

Investigations recently made by the Health Board in Manhattan show (1) that the number of bacteria in the air of streets depends on cleanliness of the streets, which in turn depends on the nature of the pavement, and (2) that many more times the number of bacteria exist at the street level than 6 feet above it. From this it follows that it is the children in the street who are especially exposed to the action of the various micro-organisms.

It is among the weaklings that the great majority of deaths occur each year, so that any sanitary measures (public or private) which tend to decrease the number of these must necessarily have a direct bearing on the diarrheal death-rate.

PUBLIC MEASURES OF PREVENTION

CLEAN STREETS

This is a possibility only to be realized by an able and honest public administration, and the first requisite to its realization is the proper paving of the streets. Uneven, broken pavements, containing numerous unpaved spaces and widely separated cobblestones, render the thorough cleaning of the streets wellnigh impossible. Vacant lots used as receptacles for all sorts of refuse and filth, and which in summer time are used, in the absence of better places, as children's play-grounds, should be properly fenced and inspected, and the necessity of regular and rapid disposal of household garbage, especially during the summer months, should be impressed upon the people.

In the borough of Manhattan this work is at present being creditably performed, and it is to its partial neglect in the neighboring borough of Brooklyn, especially in the matter of street paving, vacant lots, etc., that Ager ascribes in part a greater diarrheal death-rate in the latter borough.

TENEMENTS

It is fortunate for the cause of the poor that private philanthropy and financial sense go hand in hand in the matter of modern tenements. It is found that such properties yield a fair and certain return on the moneys invested. Personal supervision by the owners or agents of such buildings largely does away with the possibility of overcrowding and gross uncleanliness. Municipal tenement-house inspection, with rigid enforcement of the building and sanitary regulations, accomplishes the same purpose less perfectly in the poorer class of tenements.

It is reasonable to hope that with the tearing down of the vile unaired and unlighted buildings owned by grasping and irresponsible persons, thousands of which still remain, a better type of dwelling will take their place or the grounds be opened for public air spaces.

In the following table of 250 diarrheal deaths occurring in Brooklyn last summer the distribution was as follows (Ager):

		Deaths.
In tenements: Condition	poor	97
	fair	58
	good	45
Private houses		36
Institutions		14
Total of all cases		250

PUBLIC PARKS

Among the most important factors contributing to the health of city children, especially in the summer months, is the increasing number of well-kept open spaces used as recreation grounds. Here the children may spend their time instead of upon the intensely hot pavements, and mothers may take their infants from the stifling sleeping-room and the dangerous fire-escape, formerly their only way of getting air. Such spaces should be pre-empted by cities by buying up unimproved property or by tearing down undesirable tenements in all the thickly populated centers, for it is evident that in the growth of large cities there will be more reluctance on the part of municipal governments to spend the additional money required in order to condemn improved real estate.

Such parks need not be large, but they should be numerous. Large parks at great distances from the congested centers are of little use to infants and young children, for the poor have neither the time nor money to spend in reaching them. Public baths should be built in connection with these parks.

Recreation piers, such as are being used in New York to-day, fulfil the same needs. Floating hospitals have been the means every summer of saving countless numbers of children who inevitably would have succumbed to the disease under the conditions from which they were removed. Country outings, when wisely managed, are being better appreciated by the poor, and when open to mothers and infants are to be especially commended.

PUBLIC SUPERVISION OF THE MILK SUPPLY

Summer diarrhea is essentially a disease of artificially fed children. Nursing children, while not exempt, are affected much less often, and then, in the great majority of cases, by a mild form of the disease.

How often the changes produced in milk by excessive impurities are directly responsible for the severe forms of enteric disease we are not now in a position to know. That it is a very frequent cause of the digestive disturbances so often occurring in the summer months and which so frequently terminate in a more serious condition cannot reasonably be doubted.

Milk, at its best, is impure; at its worst, a little better than sewage. In the effort to obtain a germ-free milk, the medical profession began by sterilizing it, followed that by pasteurization at high temperature, then by the same process at a lower temperature, and at present I believe a majority of pediatrists are feeding raw milk, except in the summer months, when it is still considered desirable to pasteurize it, in view of the rapid changes which the bacteria produce at this season.

These changes in ideas have been brought about by the knowledge that nutritional diseases (rickets and scurvy) are much more apt to follow the constant feeding of cooked milk. To-day the cry of the profession is for milk that is clean.

Supervision by boards of health and competent physicians is doing much to improve the quality of milk. Unscrupulous dealers are summarily dealt with, and those who are willing to adhere to certain rules laid down by the milk commissions are rewarded by the enhanced value of their sales. Even the poor people are beginning to distinguish between the reliable and unreliable dealers. The County Medical Society of New York has had for several years a well-organized milk commission, composed of physicians thoroughly familiar with the subject, which is doing an inestimable amount of good in the improvement of the city supply. The expenses of this commission are quite willingly paid by the milk dealers themselves, for the reason above stated. The Pediatric Society of Philadelphia also has appointed a milk commission that is actively engaged in a crusade for pure milk, furnishing certificates to those dealers whose milk reaches the required standard of excellence and purity.

Two grades of milk are now produced under the supervision of the milk commission of New York: (1) Certified milk. For the production of this, it is required that the barnyard must be clean, free from smell, and moderately free from flies in summer. The stable must be kept clean, must be well ventilated, sufficiently lighted during the milking hours in winter, and supplied with soap, water, wash basins, and towels. The manure must be removed daily, and the stable must be kept as free as possible from dust. The water supply must be pure, the cows must be examined by a skilled veterinarian, and tested with tuberculin. They must be carefully fed, so that the flavor of the milk will not be affected. They must be groomed daily, and the udders wiped with a clean damp cloth before each milking, and, if necessary, washed with soap and water and then dried. The long hairs of the udders must be clipped, and the cows must be kept standing between the grooming and the time of milking. The milkers must be clean, and must not milk after being exposed to any contagious disease or when sick. Wet milking is forbidden. Washable overalls must be worn during milking. The milking stools must be clean. Young children, as well as cats and dogs, are excluded during milking hours. The first milk expressed from the teats must be discarded. The milk must be rapidly cooled to below 45° F., and kept cold. The dairy utensils must be thoroughly cleansed and sterilized. The dairy building must be separate from the house. The bottles of milk must be stoppered with pasteboard disks and covered with metal caps. This milk must be sent to the laboratory weekly, and is subject to examination at any time. The farms are inspected at intervals. It is required that the average bacterial content shall be less than 30,000 to the cubic centimeter. This certified milk is sold by different dealers at prices varying from 10 to 15 cents a quart.

(2) The second grade of milk is called inspected milk. This milk is designed to be produced in a cleanly and scientific manner without imposing upon the producer any expensive care which is not considered absolutely necessary. It must be of good quality, having 4 per cent. of butter fat, and it must not contain more than an average bacterial content of 100,000 in summer or more than 60,000 in winter. The barnyard must be kept clean. The stables must be well ventilated and well lighted, and they must be supplied with washing facilities for the men. The manure must be removed daily, and a sufficient number of lanterns must be supplied. The water supply must be kept pure. The cows must be kept clean. The milkers must be healthy and must not work after exposure to infectious diseases. They are required to wash their hands before milking, to use dry milking, and to put on clean overalls before milking. The dairy utensils must be kept clean. The milk must be rapidly cooled and promptly shipped. These farms are inspected by the commission's inspector and samples of the milk are examined once a month. This milk sells in New York at retail for about 8 cents a quart, the ordinary price of milk produced without sanitary supervision.[1]

Some idea of the value of the regulations laid down by the commission may be obtained from a report of Dr. W. H. Park, of New York, to whom the medical profession is indebted for the results that have followed his bacteriologic studies of milk. Park[1] states that with only moderate cleanliness on the part of the farmer—clean pails, clothes, cans or bottles, and hands, a fairly clean place for milking, and a decent condition of the cow's udder and adjacent belly—milk, when first drawn, will not average in hot weather over 30,000 bacteria to the cubic centimeter, and in cool weather not over 25,000. If these rules are neglected the bacteria will run up to 1,000,000.

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[1] From a recent report by Dr. R. G. Freeman to the New York County Medical Society, Arch. of Pediatrics, August, 1902.

New York milk in ordinary shops averages in the coldest weather over 300,000 bacteria to the cubic centimeter; in cool weather over 1,000,000, and in hot weather 5,000,000.

As showing the importance of rapid cooling of the milk and keeping it at a low temperature, the following statement from the same paper is of interest. The writer states that freshly drawn milk contains a slight and variable amount of bactericidal properties. At temperatures under 50° F. these substances act efficiently (unless the milk is filthy) for from 12 to 24 hours, but at higher temperatures this effect is very soon completely exhausted. Thus the bacteria in fresh milk, originally 5000 to the cubic centimeter, decreased to 2400 in the portion kept at 42° F. for 24 hours, but rose to 7000 in that kept at 50° F., to 280,000 in that kept at 65° F., and to 12,500,000,000 in that kept at 95° F.

Other cities in America and Europe are working along lines similar to those described. At the present writing a series of editorial articles running in the *British Medical Journal* (March 21, *et seq.*) show that the difficulties of producing clean milk are not confined to any one country or locality, and that the profession is thoroughly aroused to the importance of the subject.

MILK DEPOTS

It is quite evident that many of the people are unwilling or unable to pay the increased price for good milk, and it is to obviate this difficulty in part that a number of charitable associations and individual philanthropists, notably Mr. Nathan Strauss, of New York, have established milk depots where pure milk may be obtained at cost, either whole milk or one or two simple modifications. Those who are too poor to pay may obtain, at various hospitals and dispensaries, tickets entitling them to a day's supply, which may be renewed at the discretion of the physician.

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[1] Jour. of Hygiene, vol. i, p. 391.

EDUCATION OF THE PEOPLE

Among those who have kept in touch with dispensary pediatric practice, where a large proportion of the cases are feeding problems, it is a matter of general comment that within the last 5 years there has been a vast improvement among the poor in the knowledge of the proper care and feeding of their children. It is not long ago that artificially fed babies who were given fresh cow's milk were rarely seen in dispensaries, the vast majority of them being brought up on artificial food of various kinds. To-day nearly all the more intelligent mothers are willing to accept the advice of the physicians and take the additional trouble and time which are entailed by proper feeding. That the education of the poor is a factor of real importance in decreasing the death-rate from diarrheal diseases is shown by the statistics of Ager in the borough of Brooklyn. That the same knowledge of infant feeding among dispensary patients does not exist in the latter borough as in that of Manhattan, I can affirm from a study of a large number of cases coming from Brooklyn to attend the University and Bellevue clinic in Manhattan. This fact is doubtless due to the inferior dispensary facilities in the smaller borough. Ager finds as a result of a number of cases of diarrhea investigated last summer in Manhattan and Brooklyn boroughs, in regard to the kind of food given, the following:

	Manhattan.	Brooklyn.
Nursed	19	9
General diet	3	16
Condensed milk	27	60
Prepared food	8	5.1
Fresh milk	42	10
Goats' milk		.8

The ratio of the use of condensed milk in the two boroughs among apparently healthy babies was, in Manhattan, 1 to 4; in Brooklyn, 3 to 1. That is, 12 times as many babies are thus fed in Brooklyn as compared with Manhattan.

This the writer believes to be another important factor in causing the increased death-rate in the borough of Brooklyn; and when we consider the well-known vulnerability of infants brought up on various patent foods and condensed milk the writer's conclusions on this point cannot reasonably be doubted.

PARTICULAR MEASURES OF PREVENTIVE TREATMENT DURING THE SUMMER MONTHS

CLOTHING

This should be as light as possible. The fear of children taking cold is not confined to any particular social class. Among the rich and the poor a certain number of mothers habitually overdress their children, even in the hottest weather; but I do not recall a single case in which there were too few clothes on a healthy child. Especially is this true among the very young infants of the poor, who are generally overdressed.

AIR

For those who can afford it, the removal of children during July and August from large towns is a great advantage. For those who cannot, parks, piers, boat excursions, and country outings are the only substitutes. Theoretically, mountain air is to be recommended for dwellers by the sea, and vice versa. In my experience diarrheal affections are somewhat more frequent at the seashore than at inland resorts, a fact which may in part be explained by the occurrence of violent changes in temperature, prevalence of fogs, and atmospheric humidity on the coast, which not infrequently act as exciting causes of the milder diarrheal affections.

BATHING

Infants and young children should be frequently bathed in hot weather, especially at night-time. Nothing is more conducive to the promotion of restful sleep than this.

DRINKING WATER

This is a matter very frequently neglected. Mothers who worry so much over a child's hunger seldom think of the possibility of thirst. Water should be given with as great regularity as food, and, unless one is certain of its purity, it is better to boil it or to give instead one of the numerous bottled waters.

FEEDING

It is safe to say that a majority of infants are overfed in the summer months. If parents would stop to consider how much less food they themselves require at this time they would cease to worry over the baby when it refuses to finish its bottle or empty the breast. In this respect the baby is no doubt wiser than its parents.

While I do not believe it necessary to cut down the amount of food given to a healthy child during the summer, provided it shows no sign of indigestion, the food should not be forced, and the regular increase in the strength of the milk given to bottle-fed babies should be at a very slow rate or, better, deferred until the cool weather.

Of the food elements, an excess of fat during the hot weather is more often responsible for attacks of intestinal indigestion than sugar or proteids. Two per cent. of fat in a milk modification makes a better summer food than one containing 3 to 4 per cent. of fat. For children who are taking whole milk, fat in excess of 4 per cent. should be avoided.

TREATMENT

In every case of acute diarrhea occurring in an infant in hot weather, however insignificant it may appear, the great prime requisite of treatment is to *stop the milk,* and for the reason that there is no better culture medium for intestinal organisms. For the first 12 or 24 hours of the attack abstention from all food is most desirable, boiled water being given at regular intervals. It is most probable, in severe cases at least, that there is little desire for food at this time. And yet it is often very difficult to overcome the mother's fears of the baby starving or growing weak. If it seems desirable to give any food at this early stage, nothing does less harm than barley water. (Robinson's patent barley, 1 teaspoonful, to a pint of water and cooked for 20 minutes or more.) This may be given in small quantities, according to the child's age, at intervals of 3 or 4 hours. Rice water may be used in the same way.

This may be kept up during the whole attack, the quantity and strength of the cereal being gradually increased or other substitutes for milk given, either combined with it or separately. Among these may be mentioned egg albumin, malted milk, imperial granum, beef juice, chicken and mutton broth, liquid peptonoids, and panopeptone. The meat broths, malted milk, and egg albumin sometimes seem to increase the number of movements, and for this reason are not always desirable. Dextrinizing the cereal food is often an advantage.

As convalescence approaches and the bowel movements become fecal in character, milk may be resumed very cautiously. A teaspoonful at a time may be added to the food two or three times a day and immediately stopped when the movements show signs of increasing in number or of intestinal indigestion. The time required to get the child back on its regular diet is extremely variable; perhaps three weeks may be taken as a fair average in cases of true enterocolitis. Kerley[1] mentions one case in which the milk could not be renewed for five months without bringing on a return of the trouble.

All cases of summer diarrhea in the early stages are infection of the intestinal contents, and not until later a disease of the intestines themselves, for which reason the bowels should be thoroughly emptied at once by castor oil in full doses for those that can retain it, by calomel in divided doses ($^1/_{10}$ grain, 0.006 gram) every half hour, when there is vomiting.

DRUGS USED FOR THEIR EFFECT ON THE BOWEL

Of these, bismuth alone has stood the test of time. The subnitrate should be given in doses of 10 grains (0.65 gram) every two hours, alone or in chalk mixture or mucilage as a vehicle. Smaller doses are of no use and much larger ones are undesirable. I have found a ball of bismuth as large as a pigeon's egg in the stomach of a dead infant to whom huge doses had been administered for a diarrhea.

For reasons which I am unable to explain, in many cases of enterocolitis bismuth does not blacken the stools; in which case it appears to be useless and better discontinued. When it has been kept up, I have noticed that as the attack begins to wear away the stools will be blackened. Kerley also notes this fact, and recommends that in such cases sulphur be added to the bismuth, causing a sulphid to be formed in the stools and blackening them. I am unable to substantiate this statement.

Other salts of bismuth, preferred by some practitioners, are the subcarbonate, subgallate, salicylate, and betanapthol. They do not seem to me to possess any particular advantage over the subnitrate.

Castor oil, a few drops at a time, is combined with the bismuth by the physicians of the New York Foundling Hospital, who speak highly of its effects.

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[1] Arch. of Pediatrics, June, 1902.

Salol, ½ grain (0.03 gram), alone or with bismuth, every two hours is recommended by Koplik.[1] It has the disadvantage of upsetting the stomach.

Resorcin, ¼ to ½ grain (0.015 to 0.03 gram), in solution or combined with bismuth, chalk, and opium, and given every two hours, is highly spoken of by Jacobi.[2]

Among other remedies recommended may be mentioned various acids, notably lactic and dilute hydrochloric acid, for their supposed effect on intestinal fermentation; alkalies, lime water, and bicarbonate of sodium in vomiting and acid fermentation; salicylate of sodium, 1 to 2 grains (0.06 to 0.12 gram) every two hours for a child of a year, for its effect on persistent loose movements. It is unnecessary for me to mention more of these; their steadily increasing number is sufficient evidence of their general inutility.

SEDATIVES

Opium should be used with caution, and not at all until the intestinal tract has been thoroughly evacuated, when it is of the greatest use to quiet restlessness and diminish the intestinal peristalsis.

Dover's powder, ¼ to ½ grain (0.01 to 0.03 gram), repeated every two hours until an effect is produced; paregoric in 5 minim (0.30 c.c.) doses, and laudanum, ¼ to ½ minim (0.015 to 0.03 c.c.), are of equal merit. Hypodermatic injections of morphin, $^1/_{100}$ grain (0.0006 gram), are of especial value when medication by the mouth is contraindicated and an immediate effect desired.

STIMULANTS

These are needed in all the severe cases accompanied by marked prostration and cardiac weakness. Good whiskey and brandy stand at the head of the list and should be given in doses of 10 to 20 drops (0.6 to 1.2 c.c.) well diluted every two or three hours. In milder cases, and those in which the heart action is good and there is no marked prostration, it would seem better practice to omit the stimulants until the symptoms call for them. Instead of whiskey, or combined with it, strychnin, $^1/_{200}$ grain (0.0003 gram), and strophanthus may be used.

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[1] Diseases of Infancy and Childhood.

[2] Therapeutics of Infancy and Childhood.

In case of collapse, such as occurs in weak children and in the severe forms of the disease, hot mustard baths, heat to the extremities, and injections of hot water into the bowel are immediately indicated. In case the body fluids have been drained away by frequent movements, saline solution injected into the bowel is to a certain extent absorbed and serves to counteract this condition; but for more rapid effect, hypodermoclysis is of the greatest service. For this purpose, normal salt solution (salt 4 parts, sodium carbonate 3 parts, water 1000 parts; or 0.6 per cent. solution of common salt) may be injected into the cellular tissue of the buttocks, thighs, or back. The solution and the instruments should be absolutely sterile, and ½ pint (250 c.c.) at least injected in the course of 24 hours.

Koplik calls attention to the fact that small quantities (10 to 40 c.c.), frequently repeated, are better than the large doses at first used by Monti and which were frequently followed by internal hemorrhage. A hypodermatic needle securely fastened to a rubber tube and this connected with a Davidson syringe, as recommended by Dawbarn, answers every purpose of this operation and is readily obtained. Care should be taken to avoid the injection of air into the tissues.

ANTIPYRETICS

For the fever alone, treatment is not often required after the first 24 hours. When, however, it persists and is accompanied by restlessness and other nervous symptoms, frequent bathing is indicated. The patient should be put into a bath of a temperature of about 100° F., which should be rapidly reduced 15° or 20° by the addition of cold water. The bath may be continued for from 5 to 15 minutes, after which the child should be thoroughly dried and wrapped in blanket. A return of the symptoms calls for repetition of this procedure as often as every two hours.

Rectal irrigation with iced water carried high into the bowels and allowed to return freely is also of the greatest value, alone or alternating with the bath.

LAVAGE OF THE BOWEL

For its effect in shortening the course of the disease and emptying the bowel this is at present our most valuable remedy, and should be used in all cases in which mucus and blood continue day after day in spite of the other measures recommended. The points to be emphasized in this operation are: (1) A small rectal tube should be passed into the bowel for a distance of from 9 to 12 inches; (2) from 1 to 2 quarts (liters) of water should be used at a time; (3) the douche bag should be held 3 to 4 feet above the patient, and (4) salt solution, 1 dram to the pint, is preferable to most of the solutions recommended.

While this operation is probably without danger to life, I have seen at least two cases of collapse follow it in infants weakened by the disease. It should not be repeated oftener than once in 12 hours under any circumstances, and 24-hour intervals are to be preferred.

There are some cases in which lavage seems to increase the intestinal peristalsis and is therefore better omitted.

VOMITING

Most of these cases begin with more or less vomiting. When this persists, all medicines and food by the mouth should be stopped. Stimulants hypodermatically, food by the rectum after lavage, sedatives (especially morphin) in small doses subcutaneously and repeated until an effect is produced, and for such cases as do not yield to this treatment, stomach washing, are of the greatest value.

An important adjunct to successful treatment is fresh air. Children are to be taken into the open air as soon as possible on good days, and if their sick room be hot it is much better to move their bed when possible into the open air during the entire day.

Obviously this will not often be possible in cases occurring in the city. Careful nursing night and day, preferably by trained nurses, is of the greatest possible value.

AFTER TREATMENT

In certain cases the bowel symptoms continue with more or less severity for many weeks or even months, in spite of all we can do. For some of these cases lavage of the bowel with saline solution two or three times a week is of advantage. In other cases, especially those in which the movements more or less constantly contain small quantities of blood, indicating ulcerations of the bowel, astringent injections may be used. Silver nitrate solution (1 to 1000) in small quantities may be injected into the bowel two or three times a week. Other cases fail to respond to any treatment, and for these the one essential is a change of climate, which is often surprising in its result. Iron, arsenic, and cod liver oil should be used in convalescence.

To recapitulate, in every case of summer diarrhea: (1) Starve the patient for 12 to 24 hours. (2) Empty the intestinal tract. (3) Stop the milk until the movements are again fecal, when it should be begun very cautiously. (4) Starchy foods are the best substitutes for milk. (5) Bismuth in 10-grain (0.65-gram) doses frequently repeated. (6) Lavage of the bowel. With this treatment, under favorable conditions and in previously healthy children, the disease will terminate favorably in the great majority of cases. In weak children and in those who live in institutions and among other unhealthy surroundings, the issue is always doubtful. It is not possible at the present time to count upon treatment by a specific serum in the near future. While such a discovery is devoutly to be wished for, it is quite certain that it could only be of use in such cases as are dependent on a specific organism, or class of organisms, and that the efforts of the medical profession and the public should be continued to be directed to carrying out the various measures of prevention and treatment which I have dwelt upon at length, and which are producing such excellent results.

THE TREATMENT OF CHOLERA INFANTUM

BY A. B. MARFAN, M.D.
Physician to the Paris Hospitals

The treatment that I am in the habit of employing in cases of cholera infantum, and which you will find as successful as any in dealing with this very serious complaint, is composed of three factors: (1) abstinence from everything but water for a period of 24 to 48 hours; (2) subcutaneous injections of artificial serum, to which caffein can be added or not, according to circumstances; and (3) hot baths, either plain or with mustard flour. During the early or toxic phase of a case the patient is given no drug of any sort by the mouth, and I never prescribe washing out either of the intestine or of the stomach. I will give farther on the reasons why I refrain from the use of medicines, and from mechanical cleansing.

I

For several years now I have considered abstinence from everything but water, or what is called the *hydric diet,* as the fundamental element in treating a case of cholera infantum, and I have published the history, indications, technic, and results of this course of action on different occasions. For the present, then, I shall limit what I have to say to the bare facts that are indispensable for the practitioner.

This hydric diet, or total abstinence from everything except water, consists in preventing the child from getting any food whatever, and in giving it nothing but pure water to drink. I need hardly say that nothing but sterilized water should be given to the child. For all practical purposes, water that has been boiled for a few minutes can be considered sufficiently purified. This liquid should be preserved in the vessel in which it has been boiled, and all unnecessary change of receptacles should be scrupulously avoided. The water should be given either cold or warm, just as the child may prefer, and in a bottle or cup that has been carefully cleaned in boiling water. With some children it is necessary to administer it with a teaspoon. A certain number of physicians prefer the water from a natural mineral spring in place of boiled water; but this is not within the reach of everybody, and simple boiled water is all that is necessary to fulfil the purpose desired.

What quantity of water should be given to a child in 24 hours? At this point I must ask to be allowed to make a short digression. One of the reasons that has stood in the way of the adoption of this method, and I must admit that it caused me also a certain amount of hesitation in the early days, is that formerly we were under the impression that a very young child was unable to stand abstinence from food; we even thought that it was dangerous to deprive a baby of nourishment, if only for a very short period of time. But experience has taught us that what an infant really has difficulty in standing is much more abstinence from water than abstinence from milk. A water diet, therefore, must be based on the following precept, and I now return to my subject: *it is necessary to replace, as far as possible, the amount of milk that is not given by an equal amount of water.* Thus, to a baby five months old should be given nearly a liter of water every 24 hours. I may as well tell you that some babies do not manifest a very great degree of enthusiasm for pure water, and take to the diet with considerable difficulty; in such cases you will have to do the best you can, and get down as much water as will be feasible.

The results of this abstinence from everything except water, in cholera infantum, are altogether remarkable, and it is not difficult to see why. In the first place, and above all, *the water diet puts an end to all gastro-intestinal fermentation and putrefaction* by shutting off the supply of food to the microbes that are swarming in the digestive tract; furthermore, it gives the gastro-intestinal mucous membrane a chance to rest, and does not irritate it, as do the greater number of the stimulants and antiseptic remedies that one is tempted to administer in such cases. For these reasons, the digestive symptoms improve rapidly; vomiting stops first, and then the motions become less frequent and less liquid. Beside this, the water diet quenches the patient's thirst, which is sometimes very imperious; it prevents the dehydration of tissues, which is always very noticeable; and it keeps up and stimulates diuresis, which is so important for the elimination of toxins. In many cases, as soon as the water diet has been begun, the child, who was restless and crying, passes into a deep and refreshing sleep.

In order to obtain these good results, the water must be given pure, without the addition of any substance; it is only in the unusual cases in which children refuse to take it, or in the still more unusual cases in which the parents are alarmed at this method of treatment, that I permit, although much against my will, the addition to the water of a very small quantity of sugar or of a little weak tea. I do not allow any alcohol to be added to it, as this substance irritates the digestive mucous membrane; nor do I allow bouillon or albuminous water, which are liable to ferment and to form good culture media for microbes.

In cholera infantum, the duration of this diet should be at least 24, and not more than 48, hours. At the end of 24 hours the child should be examined to see whether it is prudent to resume the administration of food. If all vomiting has disappeared, and if the diarrhea is less, the child's appearance better, and the temperature about normal, you can authorize every four hours either a short period of nursing at the breast, or 2 ounces (60 c.c.) of sterilized milk and sterilized water (milk 1, water 2 parts). Between these feeds you should direct the use of boiled water to be continued. If this course of action seems to succeed, you can increase gradually the amount of milk, while at the same time you lessen the amount of water, and little by little you can give the feeds nearer together, until you finally get back to the child's normal diet. When, on the other hand, the symptoms have not yielded at the end of 24 hours, the water diet must be continued for another 12 or 24 hours. In any case, if the symptoms reappear when you have begun to feed again, you must not hesitate to resume the water diet for 10 or 12 hours.

II

The most striking effect of the water diet is the very rapid sedation of all the digestive disturbances. In a few hours' time the vomiting disappears, the motions become much less frequent, and their fluid character decreases; so that, if we were to judge the situation according to the digestive symptoms only, we might be led to consider the child out of danger. In this way, however, there would be many chances of our being mistaken. During the period when I relied on the water diet alone, I frequently saw the digestive symptoms disappear, or decrease considerably, under the influence of the treatment; and yet the general symptoms of the patients continued, and the infant appeared to succumb to a very rapid sort of cachexia. How can this be explained? No doubt by the fact that the origin of the gastro-intestinal toxins had been suppressed too late, and because the system was already saturated with poison from which it was no longer able to free itself. But this fatal termination has become much less common since I began to prescribe with the water diet *hypodermic injections of salt solution.* These act by stimulating the nervous system and the circulation, and by promoting diuresis, while we may consider them as powerful eliminating agents for microbic toxins.

These injections have been recommended in cholera infantum by a large number of writers, and their efficacy generally admitted. The only point that is open to discussion is whether it is best to use the solution in large or in small amounts. So far as I am concerned, I prefer to use moderate quantities. The proper solution consists of chlorid of sodium 8, and sterilized water 1000. In case of necessity this solution can be made extemporaneously by dissolving a teaspoonful of table salt in a liter of boiled water. A single injection should consist of from 10 to 30 c.c., and can be repeated according to circumstances two or three times every 24 hours. It should be made beneath the skin of the abdomen by means of a 20 c.c. syringe, such as we use for injecting antitoxin in diphtheria. The liquid should be injected very gradually, so as to avoid the formation of a large ecchymosis. This injection treatment can be continued for three or four days, and can naturally be resumed at any moment, if its indications reappear. In a general way, it is advisable not to continue it without interruption for more than five or six days; otherwise it may end by inducing considerable nervous excitement, and, later on, a sort of pale swelling of the tissues, or even actual edema.

The results produced by these injections of salt solution are perfectly manifest. A short time after they are begun, the pulse, which had been very weak and difficult to count, is found to be full and distinct; the breathing is more regular and ample, while the eye is less sunken and the expression brighter; the lips, which were a purplish-gray color, become pink; the face gradually loses its toxemic character; the extremities become warm; the central temperature of the body has a tendency to increase slightly; and, shortly afterward, urine is passed more freely and frequently. The injections have, therefore, a very favorable action on the toxic infectious condition.

I have never tried to perform intravenous transfusion of salt solution, because great difficulty is experienced in isolating the veins in very young children.

In many cases, especially when there is threatening collapse, I add a small dose of caffein to the serum, according to the following formula:

℞	Citrate (or benzoate) of caffein	12 grains	0	75
	Chlorid of sodium	40 grains	2	50
	Boiled water	10 ounces	300	

This preparation has a very powerful stimulating effect on the heart and on the nervous system.

Some physicians have made the proposition of replacing the salt water by natural serum taken from cattle or horses. It is said that this method has all the advantages of the injections of salt water, and that, in addition, it supplies the system with a certain amount of nourishment in a way other than by the stomach and intestine. This idea might be tested, but difficulty will always be found in having at hand an aseptic natural serum; whereas sterilized salt water can always be readily obtained.

III

With water diet and injections of artificial serum, I combine the use of *warm baths at* 95° *F.* (35° *C.*), of five to ten minutes' duration, given twice or four times a day. These baths are soothing to the nervous system, and are generally followed by quiet sleep; they regulate the temperature and favor diuresis. When collapse seems imminent, mustard should be added to the bath, as was recommended by Trousseau in former days; this mustard bath is a powerful stimulant to the circulation.

Since I have been using systematically the treatment indicated above, I have obtained some remarkable series of recoveries. The deadly nature of cholera infantum is too well known for anyone to think that I have merely fallen on some lucky series. Unless one is called in *in extremis,* or finds himself in the presence of one of those cases that are beyond all help from the start, there are reasons for thinking that many a child's life will be saved by this treatment. When it is used in a case of this nature, the toxic symptoms usually disappear in 24 or 48 hours, and are rarely followed by signs of typhoid prostration or by complications in other parts of the body.

IV

I must now explain why I have banished from this treatment a certain number of means that are customarily employed. Thus, surprise may be felt that I do not resort to washing out the stomach or intestine. This means, which is excellent in other forms of digestive disorder of infants, may have the most serious consequences during the toxic phase of cholera infantum. In a certain number of cases, first after washing out the stomach, and later after treating the intestine in the same way, I have observed the occurrence of very grave symptoms—at one time convulsions, at another collapse. As these accidents happened a short time (from fifteen minutes to an hour and a half) after treatment, I had no choice but to attribute to the latter some action in their origin; and although such cases are rare, still, I decided that it was preferable to give up this means of treatment. How can these accidents be explained? Here it is necessary to recall the condition of the digestive tube in cholera infantum; on the one hand its epithelium is very fragile, and on the other its contents are very toxic. Washing acts on the epithelium as a trauma, which may make its lesions worse; the water, which enters under pressure, loosens the mucus and other substances that are adhering to the walls, and may perhaps also dissolve certain toxins, and in this way render massive absorption possible. However this may be, in the acute phase of cholera infantum it is preferable not to wash out either the stomach or the intestine.

I also refrain from using any of the drugs generally prescribed for diarrhea. Thus, I give neither benzonaphthol, betol, salol, bismuth subnitrate, calomel, lactic acid, tannin, nor opium. No one of these substances has seemed to me to act favorably during the choleriform phase. I do not give alcohol, as it has often seemed to me to be more harmful than useful. As for the administration of beer yeast in infantile gastro-enteritis, I have had no experience with the subject.

V

When the toxic symptoms have disappeared, the physician's line of conduct should change, as then several different situations may arise.

When there is no typhoid state, and no complication in other organs, there remain only a tendency to loose bowels and great prostration. Convalescence in this case requires close watching. Regulation of the food is then the important point; you must know how to modify it according to the case, day by day and hour by hour.

When, after the disappearance of the choleriform symptoms, feeding is resumed, I prescribe a teaspoonful (5 c.c.) of the following preparation five or six times in the 24 hours, a little before meals:

℞	Tincture of cachou	2.5 drams	10
	Tincture of calumba	1.25 drams	5
	Bismuth subnitrate	30 grains	2
	Benzonaphthol	15 grains	1
	Mucilage		
	Water, of each enough to make	3 ounces	100

To be shaken.

If this is not followed by a noticeable decrease in the diarrhea, I prescribe tannigen—4 grains (0.25 gram)—twice or four times a day, according to age, in a little water or milk before meals. But if at any time the diarrhea becomes more profuse, and particularly if vomiting reappears, the child must be put back on a water diet for 10 or 12 hours; and if the patient is more than six months old, and a certain amount of time has elapsed since the attack began, calomel in very minute and frequent doses should be administered while the diet lasts, thus:

℞	Calomel	$\frac{1}{6}$ grain	01
	Sugar	8 grains	50

Divide into five powders, and give one every half-hour.

In short, during this convalescence without incident the treatment amounts to that of simple gastro-enteritis.

While I am on the subject of calomel, let me make the following suggestion: Do not exceed doses of $^2/_3$ grain (0.04 gram) in 24 hours in young children, and, even then, divide the dose; in this way you will obtain satisfactory results. But in the dose indicated in many text-books calomel is a dangerous drug, which may give rise to a form of colitis similar to dysentery. Calomel should hardly ever be used in children under six months of age.

When a typhoid state develops, with colitis resembling dysentery, it is the treatment of the latter complaint that you should put into effect—washing out the intestine with a solution of sodium hyposulphid (5 or 10 to 1000), mixed diet, and tepid or cool baths. In the same way, complications in other organs, such as bronchopneumonia, otitis, or cerebral symptoms, require their customary treatment. In some infants you will find that, when they have escaped from an attack of cholera infantum, a condition of chronic gastro-enteritis is left behind; in such cases you must advise the treatment of the latter disorder.

Disease of the Pancreas

THE SYMPTOMS AND TREATMENT OF DISEASE OF THE PANCREAS

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Few conditions present greater difficulties of diagnosis than do the various forms of pancreatic disease. A knowledge of their etiology, their pathology, and their relation to other diseases is doubtless essential to a clearer understanding of their symptoms. The attempt is here made to review briefly certain pathological changes affecting the gland and to point out their bearing upon clinical medicine.[1]

Disease of the pancreas is rarely recognized during life, since the symptoms, though not infrequently of great severity, present little that is characteristic upon superficial examination. The physiology of the organ being well understood it may seem at first sight surprising that disturbances of function are not more evident, but a variety of causes tend to obscure the manifestations of pancreatic disease.

Situated behind the stomach, almost in contact with the vertebral column, the organ is not palpable in health, and alterations of size comparable to those readily recognized in the liver are not discernible.

Destructive lesions, such as suppuration, hemorrhagic necrosis, and chronic inflammation, or the presence of cysts or a new growth, may so impair the functional activity of the gland that, on the one hand, it no longer furnishes the normal supply of digestive ferments, while, on the other hand, the organ may become incapable of exerting its normal influence upon carbohydrate metabolism. Alteration of the so-called external secretion is indicated by disturbance of digestion, while alteration of the internal function finds its expression in glycosuria.

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[1] For a fuller discussion of the questions involved, see "Disease of the Pancreas, its Cause and Nature," by the author. J. B. Lippincott Company, Philadelphia, 1903.

The secreting alveoli elaborate the pancreatic ferments, while the islands of Langerhans, having no communication with the pancreatic ducts, control the assimilation of sugars. Obstruction of the ducts preventing the entrance of the pancreatic juice into the duodenum causes partial destruction of the parenchyma. The chronic interstitial inflammation which results is of the interlobular type, and the islands of Langerhans are implicated only when the lesion is far advanced. In such case, although characteristic disturbances of digestion may suggest the presence of a pancreatic lesion, carbohydrate metabolism is undisturbed and glycosuria is absent. The islands of Langerhans, on the contrary, may be so altered by hyaline degeneration that diabetes results, although the secreting parenchyma is little if at all affected.

The recognition of pancreatic disease is further obscured by the fact that lesions affecting only a part of the gland may be unaccompanied by symptoms. Experimental extirpation of the pancreas is followed not only by glycosuria, but by characteristic changes in the digestion and absorption of proteids and fats. Partial extirpation of the gland is followed by diabetes only when the part remaining is a very small fraction of the whole, and experiments have shown, moreover, that a relatively small part of the gland is capable of effecting intestinal digestion. Even complete occlusion of the larger duct is not wholly uncompensated, since the smaller duct of Santorini anastomosing with the larger may, in a considerable proportion of all individuals, afford an outlet for the pancreatic secretion.

The difficulty of diagnosis is increased by the fact that disease of the organ is seldom uncomplicated, but is usually consequent upon changes in the duodenum, liver, or bile passages. Gall-stones lodged in the common bile duct, for example, are a frequent cause of both acute and chronic inflammation of the gland. A recognition of such relationship indeed may be an important aid to the diagnosis of obscure pancreatic affections.

SYMPTOMS OF PANCREATIC DISEASE

Certain symptoms, notably pain and tumor, by their localization may indicate that the organ is the seat of disease. Pain in the epigastrium is an inconstant symptom of pancreatic affections, and may be absent even when the gland is occupied by an abscess or by carcinoma. Intermittent pain resembling that of biliary colic has been observed in association with obstruction of the pancreatic duct due to calculi or other cause.

Under normal conditions the pancreas is not palpable, but in individuals with thin abdominal walls, according to Osler, particularly in women with enteroptosis, the organ can be felt. Acute or chronic inflammation rarely, if ever, causes a recognizable enlargement of the gland. A deep-seated epigastric tumor in association with acute suppurative or gangrenous pancreatitis is referable to a peripancreatic abscess limited to the lesser peritoneal cavity. Such a mass overlying the pancreas is situated behind the stomach and colon, is movable, and does not descend with respiration. A cyst or new growth occupying the gland may form a palpable mass in the same region.

It is desirable to consider: (1) The symptoms which result from impairment of the internal function of the pancreas; and (2) symptoms which follow alterations of the external secretion.

(1) *Diabetes and Alimentary Glycosuria.* — Experimental studies conducted upon a large variety of animal species have conclusively shown that complete extirpation of the pancreas is followed by a condition identical with diabetes mellitus of man. Numerous clinical observations have shown that diabetes is associated with destructive lesions of the gland in considerably more than one-half of all cases. Since the islands of Langerhans are the effective agents in regulating carbohydrate metabolism, only those lesions which affect these structures cause the disease. When, for example, the interacinar islands alone are destroyed by hyaline degeneration, glycosuria and other manifestations of diabetes mellitus are secondary to the lesion. Hence, permanent glycosuria may follow a lesion which, leaving unaffected the secreting parenchyma, is unaccompanied by any disturbance of pancreatic digestion.

Diabetes, however, may exist in the absence of any demonstrable lesion of the pancreas; doubtless the influence of the islands of Langerhans is only one of the factors which regulate the assimilation of sugars, and diabetes may be the result of changes other than those which affect the pancreas. Hence, permanent glycosuria does not give conclusive proof that a lesion of the interacinar islands exists;

but in considerably more than 50 per cent. of cases such a lesion is present. On the other hand, when a lesion of the pancreas does not implicate the islands of Langerhans, diabetes is absent, and hence it results that by far the greater number of pancreatic lesions are unaccompanied by the disease.

The attempt has been unsuccessfully made to define certain clinical symptoms characteristic of diabetes, when due to lesions of the pancreas. Lancereaux, notably, has described a special type of diabetes referable to pancreatic disease and characterized by extreme emaciation, in company with intense thirst, voracious appetite, and abundant polyuria. He designates the condition "diabète maigre," and distinguishes it from diabetes with obesity, "diabète gras," unaccompanied by pancreatic disease. His views have received some acceptance, but few clinicians maintain that it is possible to recognize the sharp distinction claimed by him. Many writers, Lepine, Hansemann, Williamson, among others, have on the one hand cited cases in which emaciation was absent, though diabetes accompanied pancreatic disease; while on the other hand in patients suffering with diabetes and emaciated they have discovered no alteration of the pancreas. Removal of the gland in animals is followed by diabetes with rapid emaciation, absence of pancreatic secretion being followed by grave disturbances of digestion and absorption. In human cases, however, diabetes may be caused by lesions of the gland which destroy the islands of Langerhans, yet leave almost intact the secreting parenchyma.

Wide-spread lesions of the organ, destroying both secreting acini and interacinar islands are accompanied by diabetes only when very little of thè gland remains. After partial extirpation in dogs diabetes does not result if more than an eighth to a twelfth be spared. For a similar reason glycosuria is rarely a symptom of acute hemorrhagic and gangrenous pancreatitis, which usually leaves relatively unimpaired a considerable part of the organ. Diffuse carcinoma attacking the pancreas may also destroy both islands of Langerhans and secreting parenchyma. Here, too, only when the lesion has almost wholly replaced the gland may glycosuria and other symptoms of diabetes be expected.

Minkowski has shown that *alimentary glycosuria* not infrequently follows partial removal of the pancreas when a considerable part still remains. Diabetes does not result, but the ability of the organ to exert its normal influence on carbohydrate metabolism is impaired, and if sugar is given to the animal in large amount temporary glycosuria results. Wille has recently observed alimentary glycosuria in association with lesions of the pancreas and thinks that this symptom may be regarded as an index to the presence of pancreatic disease. The ability to assimilate sugar was tested in 800 patients suffering with a great variety of diseases. In the morning, before other food had been taken, from 70 to 100 grams of grape sugar dissolved in a half liter of tea or coffee were administered. The urine was voided just before the sugar was taken and was then tested at intervals of two hours; when the test was positive, sugar usually appeared at the end of two hours.

Of 800 individuals upon whom this test was made, 77 subsequently died and autopsies were performed. Alimentary glycosuria had been found in 15 of those who came to autopsy, and in 10 of these grave lesions of the pancreas were present—chronic interstitial inflammation, or carcinoma of the gland, either primary or secondary to tumors in the stomach, liver, or gall-bladder. Although alimentary glycosuria may occur in the absence of pancreatic disease and is doubtless frequently associated with a variety of conditions, notably hysteria and other neuroses, chronic alcoholism, exophthalmic goiter, etc., its presence, according to Wille, suggests the existence of a lesion of the pancreas and has considerable value for diagnosis.

(2) *Disturbances of Digestion.*—Symptoms which follow impairment of the digestive or external function of the pancreas have not been very clearly defined, and though certain facts have been established, clinical observations are often divergent. The pancreatic juice, it is well known, contains ferments which aid in the digestion of proteids, fats, and carbohydrates, yet it is not the only secretion concerned in the digestion of any one of these substances. Impaired assimilation of proteids and fats is more readily recognized than alterations affecting the digestion of carbohydrates, and hence have been assigned greater diagnostic significance.

Claude Bernard destroyed the pancreas of dogs by injecting oil or other bland substance into the larger duct, and observed that the feces contained in abundance food material which had undergone little change—for example, undigested muscle-fibers or even large pieces of meat, together with fat, at times in such quantity that when cold it congealed as a layer covering the surface of the fecal mass. Subsequent observers have studied by somewhat more accurate methods the disturbances which Claude Bernard described. Abelmann found after removing the pancreas in dogs that all fat taken with the food reappeared in the feces; an exception was the emulsified fat of milk, of which about 53 per cent. disappeared. Under normal conditions little more than 5 per cent. of fat escapes absorption. Hédon and Ville claim that the statement of Abelmann is somewhat exaggerated; they found that 18 per cent. of fat which was not emulsified might undergo absorption when the gland was completely removed. After partial extirpation, even when so performed that the pancreatic juice no longer reaches the intestine, at least half the fat ingested is, they claim, absorbed. After complete removal of the organ, according to Abelmann, fat is still split, and about four-fifths of that which is unused is represented in the feces by fatty acids.

In the experiments of Abelmann after total excision of the pancreas only about 44 per cent. of the proteids ingested was absorbed, while after partial removal 54 per cent. was retained. Under normal conditions not more than 1 or 2 per cent. of nitrogenous material is unabsorbed. Loss of the pancreas also alters the normal digestion of carbohydrates, and, according to Abelmann, from 20 to 40 per cent. of starch may reappear in the feces, having undergone no transformation into sugar. Subsequent experimenters have confirmed the observations of Abelmann.

Disturbances of digestion similar to those which follow experimental extirpation of the gland have been noted in human cases when the pancreas has been partially destroyed by chronic inflammation, by cysts, or by new growths; occlusion of the duct by calculi or by tumor may have the same effect. Even when the gland is injured or its larger duct obstructed pancreatic juice in considerable amount may still reach the intestine, and in many cases digestion proceeds with no manifest impairment.

In human cases *disturbance of proteid digestion* may be recognizable by the presence of unaltered muscle-fibers in the feces. In the discharges of a patient suffering with diabetes, Fles, 40 years ago, found well-preserved striated muscle-fibers, and the administration of an extract made from the pancreas of a calf caused their disappearance. Advanced chronic interstitial inflammation with atrophy was found at autopsy. Similar impairment of proteid digestion has been not infrequently noted in cases of pancreatic disease, and in a few instances this disturbance has been tested by quantitative methods. In cases of diabetes possibly due to lesions of the pancreas Hirschfeld recovered in the feces 32 per cent. of the nitrogenous material ingested with the food. In a case of Weintraud, in which autopsy demonstrated the presence of advanced chronic interstitial pancreatitis, 45.2 per cent. of proteids ingested reappeared in the feces. A similar case is reported by Zoja.

Sahli has devised a means by which he is able, he believes, to test the efficiency of proteid digestion in the intestine. Gelatin capsules, hardened in formalin, are almost unaffected by gastric digestion but are dissolved by pancreatic juice. If such a capsule is filled with iodoform, the urine or saliva gives a reaction for iodin at the end of from 4 to 8 hours. Absence of reaction, or its delayed appearance, indicates, according to Sahli, an impairment of pancreatic digestion, provided the mobility of the stomach is normal.

As early as 1820 Kunzmann observed the presence of *fat in the feces* of a man who subsequently died with obstruction of the duct of Wirsung and induration of the pancreas, together with chronic jaundice. Fles, nearly 50 years later, described the case of a diabetic whose feces contained abundant fat; when an emulsion made from the pancreas of a calf was administered, fat disappeared from the stools. In this case no jaundice was present. A considerable number of similar cases have been described, but in many other instances of grave pancreatic disease no decrease of fat absorption has been observed. Finding jaundice associated with pancreatic disease in many cases in which fatty evacuations have indicated impaired assimilation of fat, Müller has attributed the symptom to co-existing obstruction of the common bile duct. Nevertheless, experimental as well as clinical observations leave little doubt that steatorrhea may follow loss of the pancreatic secretion.

In some cases of pancreatic disease, according to Oser, fat is discharged with the feces as an oily, yellow fluid, and the condition may be designated a true steatorrhea. In other cases the feces are clay-colored or grayish-white, and the increased quantity of fat is demonstrable only by microscopic or chemical examination; such stools contain neutral fats, fatty acids, and soaps.

An abnormally large amount of fat in the intestinal discharge is, however, by no means characteristic of pancreatic disease, and may even appear in healthy individuals after the ingestion of very great quantities of fat. The bile, it is well known, is essential to normal digestion of fats, and occlusion of the bile duct is a frequent cause of fatty stools. Moreover, the absorption of fat is prevented by certain diseases of the intestine—for example, by tuberculosis, by tuberculosis of the mesenteric glands, and even, according to Nothnagel, by extensive catarrhal inflammation and other alterations accompanied by active peristalsis. It is only in the absence of such conditions that the presence of fat is an aid to the diagnosis of pancreatic disease.

As a consequence of the impaired digestion which results when pancreatic juice is lost or greatly diminished in amount, much of the food material taken into the digestive tract passes through almost unchanged. Oser has noted the voluminous character of the fecal discharges in cases of pancreatic disease, and has emphasized the diagnostic importance of this symptom.

TREATMENT OF PANCREATIC DISEASE

In treatises on disease of the pancreas, such as those of Friedreich, Oser, and Lancereaux, are mentioned a variety of drugs which have been claimed to exert an influence upon the diseased gland, but the writers have no faith in the efficacy of these agents. Alkaline carbonates, pilocarpin, preparations of iron, of bismuth, and of mercury, particularly calomel, have been believed to produce beneficial effects when disease of the pancreas was supposed to exist. The difficulty with which pancreatic disease is recognized presents an obstacle to the establishment of an empirical therapy, and the evidence in favor of these remedies has little weight.

Experimental demonstration that diabetes results when the pancreas is removed raised hope that the disease might be successfully treated by the administration of pancreatic tissue obtained from lower animals or by the use of extracts prepared from it. The remarkable results which have followed the use of thyroid extracts in myxodema—a disease dependent upon destruction of the thyroid gland—have suggested such a possibility. A number of observers, Minkowski, Thiroloix, and others, soon found that administration of fresh pancreas and of extract to animals deprived of their pancreas failed to influence the assimilation of sugar, whether given by mouth or injected below the skin, into the veins, or into the peritoneal cavity. No better success has been obtained in the treatment of diabetes mellitus in man, although in a considerable number of cases similar methods have been employed. A few writers have claimed that some improvement followed the administration of the fresh gland by the mouth or the subcutaneous injection of extracts, but the influence upon glycosuria which they note has been temporary and inconstant, and may be referred to other causes. Hector Mackenzie, Fürbringer, P. W. Williams, Hugounencq and Doyon, Oser, and others, have found glycosuria uninfluenced by the same methods. Attempts to graft pancreatic tissue obtained from animals below the skin of patients suffering with diabetes have been unsuccessful.

Better results have in a few instances attended *efforts to supply by artificial means a deficiency of digestive ferments* consequent upon disease of the pancreas. In animals it has been found possible by the administration of fresh pancreas to increase the assimilation of proteids and fats impaired by extirpation of the pancreas. Abelmann found that the pancreas of the pig fed to dogs from which the gland had been removed aids the absorption of fat, and so favors the digestion of proteids that 78 per cent. is absorbed instead of 44 per cent. These observations have been confirmed by Sandmeyer, Rosenberg, and others.

The remarkable case of Fles, quoted by Friedreich, has already been mentioned; disturbed digestion of proteids and fat was associated with diabetes mellitus. The fresh pancreas of a calf was rubbed in a mortar with six ounces of water and the mixture strained. A part of the milky fluid obtained was taken after each meal, so that one pancreas was consumed daily. Although the patient continued upon his previous diet of bacon and fat meat, at the end of two days all fat had disappeared from the feces and the number of undigested fibers was greatly diminished. Whenever the administration of the infusion was discontinued fat and muscle-fibers reappeared. Although glycosuria persisted unchanged, the general condition of the patient improved for a time, but death finally occurred as the result of phthisis. The pancreas was the seat of advanced sclerosis.

In a case recorded by Langdon-Downs fatty diarrhea was controlled by the use of pancreatic extract; the urine contained a trace of sugar, but the condition of the pancreas could not be learned with certainty. Oser describes a case in which a tumor mass felt in the epigastrium was accompanied by jaundice; in the feces undigested muscle-fibers and fat were found. A diagnosis of carcinoma of the pancreas was made. The administration of pancreatin (Merck), 1 gram every day, taken in divided doses, was followed by well-marked improvement in the digestion of fat; the patient felt stronger.

The few cases cited suggest that pancreatic extracts may be successfully used when there is evidence that digestion suffers from deficiency of the pancreatic secretion. When the lesion is caused by malignant growth or is associated with diabetes, although permanent benefit cannot result, it may be possible to strengthen the patient and retard emaciation.

CLINICAL SIGNIFICANCE OF FAT NECROSIS

The lesion known as focal fat necrosis has been shown to be caused by penetration of the secretion of the pancreas into the tissues about the organ and results when the fat-splitting ferment of the pancreatic juice comes into contact with the living fat tissue. Occlusion of the pancreatic duct favors the dissemination of ferments elaborated by the gland, and wide-spread necrosis implicating the subcutaneous and pericardial fat as well as that of the abdomen follows ligation of the pancreatic ducts in the cat. In human cases widely disseminated fat necrosis seldom results from occlusion of the pancreatic ducts. Obstruction occurs gradually and is associated with chronic interstitial inflammation which apparently retards the diffusion of the pancreatic secretion, so that minute foci occur only in the immediate neighborhood of the gland.

Disseminated necrosis of fat in most instances accompanies acute hemorrhagic pancreatitis, or its later stage, so-called gangrenous inflammation. I have shown elsewhere[1] that the lesion is in many, if not in all, cases caused by a gall-stone lodged at the duodenal orifice of the diverticulum of Vater. Not only is the outflow of pancreatic juice prevented, but bile is injected into the pancreatic duct by the gall-bladder. As a result necrosis of the pancreatic parenchyma ensues, and whatever pancreatic secretion may be present, forced backward into the tissues about the organ, causes necrosis of the surrounding fat. The lesion more rarely accompanies suppurative inflammation of the gland.

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[1] Bulletin of the Johns Hopkins Hospital, 1901, xii, 182.

By some writers fat necrosis has been described as a disease entity associated with certain more or less ill-defined symptoms. The condition, on the contrary, is always a consequence of pancreatic disease and bears much the same relation to lesion of the pancreas as does jaundice to hepatic disease. Its presence gives evidence that the pancreatic secretion has been diverted from its normal channels into the interstitial tissue of the organ and into the neighboring fatty tissue.

In a case cited by Hansemann, foci of necrosis occurred in the subcutaneous tissue, and their presence was marked by circumscribed injection of the overlying skin. Rarely, however, does the lesion affect adipose tissue outside of the abdomen, and during life is recognizable only when the peritoneal cavity is opened. Since in every instance disseminated fat necrosis indicates the existence of grave disease of the pancreas, its recognition is of importance to the surgeon who opens the abdominal cavity. Particularly is this true when pancreatic disease has been unsuspected and operation has been undertaken, perhaps with the purpose of relieving intestinal obstruction suggested by the symptoms. With few exceptions disseminated focal necrosis encountered at operation gives evidence that the pancreas is the seat of acute hemorrhagic inflammation or of secondary gangrenous change.

Foci of necrotic fat are very conspicuous by reason of their homogeneous, opaque, yellowish-white color, in marked contrast to the surrounding translucent yellow fat, and when the omentum is exposed it is unlikely that they will be overlooked if present. To one who has seen the lesion it is unmistakable, but to the inexperienced miliary tubercles or carcinomatous nodules may be suggested by the shape and color of the sharply defined areas. The lesion, however, is not elevated and nodule-like, but on examination is found to represent a transformation of the pre-existing fat.

When the outflow of bile is hindered and jaundice occurs, biliary pigments make their appearance in the urine. Abundant evidence has shown that fat necrosis is due to the action of the fat-splitting ferment secreted by the pancreas, and this ferment has been found by Flexner in the areas of necrosis. The possibility suggests itself that the ferment which is free in the tissue may be excreted by the kidneys, though it is not improbable that the acidity of the urine may partially or completely destroy its activity.

I have examined the urine in one instance of acute hemorrhagic pancreatitis, and the result, though not conclusive, is cited in order that its accuracy may be further tested. The method employed is that described by Castle and Loevenhart: ethyl butyrate carefully purified, when acted upon by a fat-splitting ferment, is decomposed with the formation of butyric acid, which gives an acid reaction to the solution.

Following an operation at which acute hemorrhagic pancreatitis with fat necrosis was recognized, the patient voided no urine, and in the bladder at autopsy only a small quantity was found. After adding a few drops of litmus solution, this was neutralized with potassium hydroxid (one-tenth normal solution) and divided into two parts. To one part was added a few drops of ethyl butyrate. The second part, used as a control, was boiled in order to destroy the ferment, if present, and ethyl butyrate was added. Both specimens were kept at 37° C., and at the end of 24 hours the unboiled specimen had acquired a well-marked acid reaction, while the control specimen was unchanged. Owing to the small quantity of urine obtained it was not possible to repeat the test.

Recognition in the urine of a ferment derived from the pancreas would afford a certain means for the diagnosis of obscure pancreatic affection, accompanied by fat necrosis, notably acute hemorrhagic pancreatitis. Hence it is desirable to apply in suspected cases the test just described in order if possible to establish its value.

SYMPTOMS OF ACUTE HEMORRHAGIC PANCREATITIS

The difficulty of diagnosis characteristic of disease of the pancreas is well illustrated by acute hemorrhagic pancreatitis, yet the clinical picture which it presents is often distinctive, and in many instances recognition of the condition is possible.

This lesion, in a large proportion of cases at least, is a consequence of cholelithiasis and is caused by the action of bile diverted into the pancreatic duct by a small calculus lodged at the duodenal orifice of the diverticulum of Vater. Wide-spread necrosis of the parenchyma ensues and is associated with abundant hemorrhage into and about the organ. Inflammatory changes rapidly follow, and death frequently occurs within 48 hours of the onset of the symptoms. In a considerable number of cases the individual survives, and the necrotic pancreas, becoming black and softened, assumes the appearance described as gangrenous, so-called gangrenous pancreatitis representing a like stage of the hemorrhagic lesion. Bacteria invade the dead tissue, and inflammation of the adjacent peritoneum not infrequently results in the formation of an abscess cavity limited to the lesser peritoneum.

Fitz describes as follows the symptoms of acute hemorrhagic pancreatitis: "It begins with intense pain, especially in the upper abdomen; soon followed by vomiting, which is likely to be more or less obstinate, and not infrequently by slight epigastric swelling and tenderness, with obstinate constipation. A normal or subnormal temperature may be present, and symptoms of collapse precede, by a few hours, death, which is most likely to occur between the second and fourth days."

Since the lesion is caused by the lodgement of a gall-stone at the duodenal orifice of the bile duct, symptoms of biliary colic may precede those of the pancreatic lesion and may be confounded with them. The intensity of the pain, its diffuse epigastric character, with occasional localization on the left side, and the profound collapse usually present, according to Thayer, give sufficient evidence that the pancreas is implicated. Acute hemorrhagic pancreatitis is frequently mistaken for intestinal obstruction, but the intensity and localization of the pain, the severity of the collapse, and the absence of stercoraceous vomiting in most instances serve to characterize the condition.

The profound collapse and rapidly fatal termination of many cases of acute hemorrhagic pancreatitis suggest the presence of an acute intoxication. A similar condition follows the lesion produced experimentally in dogs, and death within 24 hours may be caused by the injection of 5 c.c. of bile into the pancreatic duct. Bile, doubtless in association with the pancreatic secretion acting upon the substance of the gland, produces, both in the human and in the experimental cases, substances which, when absorbed, exert a profoundly poisonous action. Such an explanation of the fatal result appears more probable than that which seeks to refer the symptoms of collapse or shock to an obscure lesion of the sympathetic ganglia near the pancreas.

In many instances the symptoms of onset are much less severe than those just described, and, perhaps associated with an attack of gall-stone colic, may be so slight as to be unrecognizable. The patient survives and the disease pursues a more or less chronic course. The symptoms are essentially those previously mentioned, but they occur with less intensity. Pain is localized in the epigastrium, and vomiting may recur at intervals. Symptoms indicative of suppuration finally give evidence that the necrotic tissue of the pancreas has become infected; there is an irregular temperature, and leukocytosis suggests the presence of an abscess. An ill-defined tumor felt above the umbilicus gives more positive proof that fluid has accumulated about the pancreas in the lesser peritoneal cavity. The appearance of such a mass in the epigastric region is due, not to the enlarged pancreas, which is rarely if ever palpable, but to the presence of hemorrhagic or purulent fluid about the organ.

Those disturbances of digestion which have been noted in describing the general symptomatology of pancreatic disease may occur, but rarely is there such complete destruction of the pancreas that its functional activity is wholly destroyed. In two cases in which I found acute hemorrhagic pancreatitis at autopsy the patients passed clay-colored stools.

In only 2 of the 41 cases of hemorrhagic pancreatitis, and in but 3 of 40 cases of gangrenous inflammation collected by Körte, was sugar present in the urine. The following interesting case of diabetes consequent upon acute hemorrhagic pancreatitis has been recently described by Franke. The patient, a man aged 42 years, who had previously enjoyed good health, was attacked 12 days before his death with severe abdominal pain, accompanied by vomiting. The pain, which was localized in the region of the umbilicus, continued, and the patient complained much of thirst. When seen, 8 days after the onset of symptoms, sugar was found in the urine, and to the amount of 3.5 per cent. was present until death. The fat below the parietal peritoneum, the fat of the omentum, of the mesentery, and of the mediastinum as well, contained numerous foci of necrosis. The pancreas was represented by a large blackish-red mass, which consisted in great part of altered blood, but in a few places still preserved a lobular structure. The gall-bladder was distended and contained a calculus about the size of a plum.

Acute hemorrhagic and gangrenous pancreatitis being caused by the lodgement of a gall-stone in the diverticulum of Vater, patients suffering with this disease not infrequently give a history of preceding gall-stone colic. In a case which I have examined the patient underwent an attack of jaundice six months before his fatal illness. In other instances there is a history of repeated paroxysms of severe abdominal pain, doubtless due to the passage of calculi which have failed to lodge at the orifice of the bile duct. Preceding attacks of so-called gastric or gastroduodenal dyspepsia, noted by Fitz, in some cases at least admit of a similar explanation. In some reported cases recurrent pancreatitis has been described, but attacks of severe pain and vomiting preceding the fatal illness are doubtless often referable to cholelithiasis. Nevertheless, it cannot be denied that acute pancreatitis may recur, for should the patient survive a primary attack, the structure of the diverticulum of Vater being favorable, a second might follow the expulsion of another stone of appropriate size.

TREATMENT OF ACUTE HEMORRHAGIC AND GANGRENOUS PANCREATITIS

What has been said concerning the etiology and pathology of acute inflammation of the pancreas demonstrates the futility of medical treatment directed to the palliation of the lesion. At the onset of symptoms the attention of the physician will be directed to the profound collapse, and efforts will be made to strengthen the enfeebled circulation. The intense abdominal pain is often uncontrollable by the use of morphin. For the continued vomiting Körte recommends that the stomach be washed out, food by the mouth discontinued, and nutrient and stimulating enemas resorted to.

It is only by surgical means that the seat of the disease can be reached. When it is possible to make a diagnosis of acute hemorrhagic pancreatitis from the symptoms which in a certain number of cases are sufficiently characteristic, the advisability of operation presents itself for consideration. During the primary collapse little is to be gained by interference which would further impair the resistance of the patient; hemorrhage is rarely profuse and bears little relation to the severity of the condition. Should the individual survive the early symptoms of shock, infection of the gangrenous tissue and abscess formation limited to the lesser peritoneal cavity soon demand exploration and drainage. Septic fever, leukocytosis, and the presence of a palpable mass in the epigastrium indicate the necessity of operative interference.

In some cases symptoms are so indefinite that acute hemorrhagic pancreatitis is not suspected, while in other instances, though such a lesion suggests itself, the evidence is insufficient to exclude the presence of other conditions. Experience has shown that the disease is often first recognized by the presence of fat necrosis noted at operation undertaken for the relief of a supposed intestinal obstruction or perhaps performed for the purpose of exploration demanded by continued symptoms of grave abdominal disorder.

Should disseminated fat necrosis indicate the presence of a pancreatic lesion, exploration of the lesser peritoneal cavity through the gastrocolic omentum may disclose the presence of a pancreatic or peripancreatic abscess and permit its evacuation. Thick abdominal walls and omental fat in large quantity, together with an accumulation of necrotic tissue and partially clotted blood within the lesser peritoneal cavity, at times make access to the pancreas itself difficult or impossible, at least undesirable.

During whatever stage of the disease operation is performed the condition of the bile passages is important and may offer an imperative indication for interference. The common bile duct should be examined so far as it is possible, and bearing in mind the mechanism by which a small calculus may produce the lesion, the operator should, if feasible, exclude the possibility that a stone is still lodged in the diverticulum of Vater. If such impaction should be found, removal of the calculus is essential in order to prevent further destruction of the pancreas. The temporary lodgement of a calculus within the diverticulum may produce an extensive pancreatic lesion, yet finally expelled into the duodenum, the stone may be no longer demonstrable either at operation or at autopsy. Hence in a considerable number of cases the gall-bladder will be found to be filled with gall-stones, even though the bile ducts are free. These stones may be of such size that any one of them lodged at the orifice of the common duct might divert bile into the pancreatic duct.

In a patient with acute pancreatitis and fat necrosis, operated upon by Dr. Howard A. Kelly, recovery followed the removal of a large number of small gall-stones from the gall-bladder. Dr. Kelly has kindly permitted me to add the following notes of this case, which will be published in detail by Dr. Louis Lehr:

Mrs. W., aged 41 years, was admitted to the care of Dr. Kelly, May 17, 1902. For several years preceding, at intervals of almost every 2 months, she had suffered with attacks of pain in the epigastrium. Her present illness began one week before with severe pain in the left side and back, lasting about 3 days, and subsequently recurring in paroxysms, necessitating the use of morphin. There was slight nausea, but no vomiting. The stools were clay colored and of very foul odor; the temperature varied from 98° to 100° F. An operation was undertaken on the tenth day of her illness. After an incision had been made through the thick abdominal wall, the omentum was found to contain opaque, white foci of fat necrosis. An oblong mass about 9 cm. in length and 6 cm. broad lying behind the greater curvature of the stomach occupied the position of the pancreas. Further examination was not possible on account of the great quantity of fat. The gall-bladder was found partially collapsed about a large number of small gall-stones. An incision was made through the skin and abdominal wall opposite the gall-bladder; the organ was stitched to the edges of the wound and opened. About 50 small calculi of almost uniform size were removed. The wound opposite the gall-bladder healed slowly, and the patient made an uneventful recovery.

In the preceding case acute hemorrhagic pancreatitis and consequent fat necrosis was doubtless caused by the passage of a small calculus similar to those so numerous in the gall-bladder. The clinical history makes it probable that similar calculi had been repeatedly expelled previous to the attack for which operation was performed, but impaction in the diverticulum of Vater had failed to occur. Removal of those still present in the gall-bladder offered the best assurance of immunity from subsequent attacks.

SYMPTOMS OF CHRONIC PANCREATITIS

Chronic pancreatitis is rarely accompanied by such definite symptoms that its recognition is possible during life. In most instances a considerable part of the parenchyma is undestroyed, and is capable of performing, in part at least, the functions of the gland. Hence chronic pancreatitis may be unaccompanied by glycosuria or by any disturbance of digestion. Since chronic pancreatitis usually accompanies grave disturbances of the stomach, duodenum, or bile passages, symptoms of the primary disorder are predominant and hinder recognition of the pancreatic lesion. On the other hand, disease of neighboring organs may suggest the presence of chronic pancreatitis.

From the clinical stand-point it is desirable to distinguish between (1) an interlobular and (2) an interacinar type of chronic interstitial pancreatitis. The first is rarely accompanied by diabetes mellitus; the latter, affecting both secreting parenchyma and interacinar islands, except in its earliest stage, is associated with diabetes.

The etiology of chronic interstitial pancreatitis has been elsewhere [1] discussed in detail. The disease is uncommon in early life, and two-thirds of the cases occur between the ages of 40 and 60 years. Chronic pancreatitis follows a variety of readily recognizable conditions affecting other organs, and hence its existence may be not infrequently suspected.

(1) *Chronic pancreatitis of the interlobular type* in the greatest number of cases is caused by obstruction of the duct produced by pancreatic calculi, by biliary calculi, or by tumors and cysts compressing the duct as it passes through the head of the gland. In many such cases disturbances of digestion consequent upon loss of the pancreatic secretion have been noted. Acute and chronic inflammation of the stomach and duodenum on the one hand, and of the bile passages on the other, may, as in cases previously described, be accompanied by interlobular pancreatitis, the result of an ascending infection. I have described 4 cases in which chronic interlobular inflammation was found at autopsy in individuals who had suffered with persistent vomiting, caused doubtless in 3 cases, presumably in all, by lesions of the stomach or of the duodenum.

Interlobular pancreatitis is accompanied by diabetes only when the lesion is so far advanced that dense sclerotic tissue, which replaces the secreting parenchyma, surrounds and compresses the islands of Langerhans. Glycosuria was noted in only 1 of my 22 cases. In this case chronic interstitial pancreatitis was caused by calculi obstructing the pancreatic ducts; glycosuria was of mild type, and disappeared when the patient was given a diet poor in carbohydrates.

In at least 5 of 15 cases in which Wille found alimentary glycosuria, chronic inflammation—from his description presumably of the interlobular type—was found at autopsy. It is not improbable that, even though the lesion does not implicate the islands of Langerhans, chronic inflammatory changes in their neighborhood may slightly impair their functional activity, perhaps by interfering with their blood supply. Where chronic pancreatitis is suspected, the presence of alimentary glycosuria, tested by the administration of sugar, would give additional evidence.

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[1] Amer. Jour. Med. Sci., 1902, cxxiii, 845.

(2) Distinctive of *the interacinar form of chronic pancreatitis* are glycosuria and other symptoms of diabetes mellitus. Diabetes mellitus, however, is caused by other lesions of the pancreas, and indeed may occur in the absence of pancreatic disease. In 9 of 19 cases of diabetes[1] which I examined at autopsy interacinar pancreatitis occurred; in 6 of these cases hyaline changes were also present. Although no group of symptoms, as Lancereaux has claimed, is characteristic of diabetes caused by pancreatic lesion, conditions associated with the disease may give evidence that it accompanies interacinar inflammation of the gland.

In a considerable number of cases described in the literature arterial sclerosis has been associated with chronic interstitial inflammation of the pancreas. In one of the cases included in my series advanced arterial sclerosis in a diabetic was followed by gangrene of the legs, and at autopsy chronic interacinar pancreatitis was found. When advanced arterial sclerosis accompanies diabetes mellitus, the latter disease is with much probability referable to interacinar inflammation of the pancreas.

The association of diabetes mellitus and cirrhosis of the liver has been very frequently observed, and in some of these cases examined at autopsy the pancreas has been found to be the seat of chronic inflammatory changes. Chronic inflammation of the pancreas and of the liver is doubtless in such cases dependent upon the same etiological factors. In 2 of my cases diabetes and atrophic cirrhosis coexisted, while at autopsy chronic pancreatitis of the interacinar type was demonstrable. I am indebted to Dr. Libman, of New York, for notes upon a similar case. These observations agree with those of Lefas, who found diffuse chronic inflammation of the pancreas in association with atrophic cirrhosis of the liver. Diabetes with cirrhosis of the liver is with much probability indicative of interacinar pancreatitis.

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[1] Disease of the Pancreas, loc. cit.

One form of chronic interacinar pancreatitis with diabetes and cirrhosis may be readily recognized. Diabetes with hemochromatosis, the so-called bronzed diabetes, is doubtless in all cases the result of chronic interacinar inflammation affecting the pancreas and implicating the islands of Langerhans. The combination of symptoms in such cases is characteristic. Diabetes mellitus is associated with hypertrophic cirrhosis of the liver, and there is often intense pigmentation of the skin. When doubt exists as to the character of this pigmentation, iron may be demonstrated by appropriate methods in sections made from a bit of skin, if such is obtainable from the patient. In a case described by Anschütz, fatty stools gave evidence that the secretion of the pancreas was diminished in amount; but even in the absence of such symptoms, diabetes accompanying hypertrophic cirrhosis with pigmentation may be attributed to chronic pancreatitis of the interacinar type.

PANCREATITIS AS A COMPLICATION OF OTHER DISEASES

It has been repeatedly stated that in most instances both acute and chronic inflammation of the pancreas are secondary to disease of other organs. Acute or chronic inflammation may be the result of an ascending infection of the pancreatic ducts having its origin in the inflamed stomach, duodenum, or bile passages. The occurrence of chronic inflammation in association with persistent vomiting has been emphasized. The close relationship between disease of the liver and of the pancreas is especially noteworthy, chronic inflammation of the liver and pancreas, doubtless due to the same etiological factor, not infrequently occurring in conjunction. Much more common, however, is the association of cholelithiasis and disease of the pancreas, and it is perhaps desirable to consider briefly the conditions under which cholelithiasis is accompanied by pancreatic disease.

Whenever a gall-stone is expelled from the common bile duct, temporary occlusion of the pancreatic duct results. I have described in detail the mechanism by which a small gall-stone lodged at the orifice of the diverticulum of Vater diverts bile into the pancreatic ducts and causes hemorrhagic pancreatitis;[1] a variety of conditions must be fulfilled in order that acute pancreatitis follow the passage of a calculus, and in only a small proportion of cases is gall-stone colic accompanied by acute disease of the pancreas. In not more than one-third of all individuals are the anatomical peculiarities of the diverticulum of Vater such that a calculus could occlude its duodenal orifice, yet fail to obstruct the bile or pancreatic ducts as they enter the diverticulum. Even should the diverticulum be of considerable length and provided with a narrow duodenal orifice, a calculus could transform the two ducts into a continuous channel only if it be of very small size. In many instances the offending calculi have not been larger, roughly speaking, than a pea. Such facts help to explain the infrequency of acute hemorrhagic pancreatitis when compared with the frequency of cholelithiasis. The symptoms which characterize acute hemorrhagic pancreatitis and distinguish it from uncomplicated biliary colic have already been mentioned.

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[1] Disease of the Pancreas, loc. cit.

A large gall-stone lodged in the terminal part of the common bile duct, or indeed within the diverticulum of Vater, prevents the escape of pancreatic secretion (unless the duct of Santorini affords a free outlet) and chronic interlobular inflammation of the gland results. The clinical importance of this condition lies in the fact that the indurated gland felt during an operation undertaken for the removal of gall-stones in the common duct has been frequently mistaken for malignant growth. Recognition of the relation between cholelithiasis and chronic pancreatitis will have an important influence upon the prognosis in such cases, and removal of the occluding calculus will prevent further development of the pancreatic lesion.

THE DIAGNOSIS AND SURGICAL TREATMENT OF DISEASE OF THE PANCREAS

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THE diagnosis of pancreatic disease is attended with such doubt and difficulty in the minds of many physicians, especially those without hospital facilities for observing large numbers of abdominal lesions, that a detailed discussion of the differential diagnosis of affections of the pancreas is of some value.

The anatomic position of the pancreas, its depth from the surface, its relation to both peritoneal cavities and to such important organs as the stomach, liver, biliary tracts, and intestines, directly contribute to the difficulties in diagnosis. The little-known character of the internal secretion of the pancreas and the relation of the islands of Langerhans to the body metabolism further complicate the diagnostic problems. We must, therefore, fall back upon such facts as have been noted in the reported cases of pancreatitis.

Roughly speaking, pancreatitis may be divided into, (1) acute pancreatitis—hemorrhagic, suppurative, and gangrenous; and (2) chronic indurative or interstitial pancreatitis.

ACUTE PANCREATITIS

ETIOLOGY.—The etiology of *acute hemorrhagic pancreatitis* is rather obscure. Flexner has shown that hemorrhage into the gland is common in all forms and may remain limited to the interlobular septa, but when excessive with macroscopic evidence of hemorrhage the blood is found between or within the acini. In these cases the pancreas is dark and swollen, with a mottled red and black appearance on section. It crumbles under pressure and is greasy to the touch. The surrounding fat, as a rule, is necrotic and may be stained a blackish color. Fat necrosis may exist in the mesentery, in the subcutaneous fat, or even in the epicardial and mediastinal fat.

The cause of this hemorrhage is not known. Whether a mechanical or chemical irritant induces inflammation followed by hemorrhage, or whether the hemorrhage is primary and the inflammation secondary, is open to doubt.

Flexner[1] found hemorrhagic pancreatitis, with rapidly fatal results, to follow injections of acids, alkalies, formalin, and bacterial suspensions into the ducts and gland. In many of these experiments it was conclusively proved that the presence of micro-organisms was not necessary to produce the hemorrhagic lesion or even necrosis of the gland, but that these may be due to the action of the infiltrating blood upon the parenchyma, with granulation and necrosis of the cells.

The association of gall-stones with disease of the pancreas has been frequently noted, and the calculi have been considered a factor in the production of acute pancreatitis. Halstead's case was of this nature and has been widely quoted. In this instance a minute calculus was detected completely plugging the opening into the duodenum, with bile in the pancreatic duct. It was believed that the sudden stoppage of the biliary flow by the impacting stone was sufficient to drive the bile into the pancreas, inducing a rapid and fatal inflammation of that organ. Opie,[2] experimenting upon dogs, found that injection of bile into their pancreatic ducts after opening the duodenum caused necrosis and hemorrhage of the parenchyma of the pancreas, followed by an inflammatory lesion resembling that in man. Fat necrosis was also present.

Hlava[3] injected Bacillus coli communis, Bacillus lactis aerogenes, and Bacillus capsulatus of Friedlander, with the production of hemorrhage and necrosis of the pancreas and fat necrosis. While believing that the products of such bacteria caused the necrosis, he was unable to produce this result by the injection of bacterial toxins.

Suppurative pancreatitis, or abscess of the pancreas, is due to infection by bacteria, is often independent of hemorrhage, and is caused either by an ascending infection from the duodenum or through the circulation. The pus is found as small abscesses, distinguishing this form from the hemorrhagic disease with infiltration and diffusion of the leukocytes throughout the tissues. Of course, hemorrhage may be coincident and the two forms co-exist.

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[1] Contributions to the Science of Medicine, Dedicated to William H. Welch, 1900, p. 743.

[2] Bull. of Johns Hopkins Hosp., vol. xii, 1901, p. 182.

[3] Quoted by Flexner, loc. cit.

Gangrenous pancreatitis, like gangrene of any other organ, is dependent upon disturbances in the blood supply and the presence of putrefactive micro-organisms. Flexner believes that this form represents a condition superadded to one of the other forms, more often following the hemorrhagic than the suppurative variety.

Oser [1] describes necrotizing pancreatitis, in which the blood collections in the hemorrhagic form may induce minute areas of necrosis with karyolysis and disintegration of the cells, and later the usual picture of coagulation necrosis with sequestration of the necrotic tissue.

Reference has been made several times to *fat necrosis,* a most interesting chemical reaction which results from the action of steapsin upon living adipose tissue. Balser, in 1882, gave a classical description of this phenomenon, and Langerhans, in 1890, by animal experimentation, proved the dependence of fat necrosis upon disease of the pancreas. He also showed the chemical reaction which took place whereby the fat was split up into a fatty acid and glycerin, the former uniting with calcium salts in the cells and the latter becoming absorbed.

Hlava, Körte,[2] Oser, and Flexner, by injecting acids, alkalies, gastric juice, and suspensions of bacteria into the ducts and substance of the gland, produced fat necrosis, together with the pancreatic lesion described earlier in this paper.

Opie [3] made a careful study of disseminated fat necrosis by simply ligating the pancreatic ducts and causing widespread necrosis of the abdominal fat as well as the fat in the subcutaneous and pericardial tissues. The pancreas itself suffers little injury, the necrosis depending upon the diffusion of the fat-splitting ferment.

Flexner showed that the fat-splitting ferment, lipase or steapsin, is not present in normal fat, while Kastle and Loevenhart[1] demonstrated it in all the tissues of the body.

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[1] Nothnagel's Spec. Path. und Ther., vol. xviii, 1898.

[2] Berliner Klinik, 1896, No. 102.

[3] Contributions to the Science of Medicine, Dedicated to William H. Welch, 1901, p. 859. Also, Disease of the Pancreas, 1903.

Recently, Wells[2] saturated bits of cotton with commercial "pancreatin," which he found as active as the fresh extract, fastened them to the omentum and studied the entire sequence of events by removing the necrotic foci at intervals. He found the process to be due to a ferment, inhibited at 55° C. and destroyed at about 71° C.; but the nature of this ferment he could not determine, and he thought that the necrosis was primary, while the fat-splitting, due to the lipase, was merely secondary to and not a cause of the necrosis. He believes that the dissemination occurs through the lymphatic system.

SYMPTOMATOLOGY.—As far as we are able at the present time to say, the history of a case of *acute hemorrhagic pancreatitis* will present evidence of previous attacks of gastro-intestinal disease, either as a result of chronic gastritis or of alcohol. In many cases the patient has experienced, or is suffering from, attacks of pain referable to the biliary tract—either gall-stones or a chronic catarrh.

The attack, in the majority of cases, begins suddenly, with severe epigastric pain, nausea, vomiting, great rapidity of the pulse, dyspnea, and cyanosis, followed by a rapid loss of strength. The early symptoms strongly resemble those of intestinal obstruction, and the exhaustion and collapse are frequently so severe as to induce death in a few hours or several days. The disease soon resolves itself into peritonitis of the upper portion of the abdomen, and in the less severe type of cases becomes subacute or eventually subsides. On the other hand, if death does not take place from exhaustion, an infection with pyogenic micro-organisms may induce suppuration or necrosis. Bilateral and nearly board-like rigidity is present from the onset. Tympany develops in the epigastrium from paresis of the stomach and intestine, but not to the extent, in all instances at least, of obscuring the liver dulness. Tenderness to pressure is pronounced early in acute pancreatitis.

The pain is colicky in character and there are either intervals of relief or else continued pain with exacerbations of severe colicky attacks. The pain is frequently increased by the taking of food.

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[1] Chem. News, 1901, lxxxii, Nos. 2150–2155.

[2] Jour. Med. Research, vol. ix, No. 1, 1903.

Constipation from paresis is the rule early and adds to the liability of mistaking pancreatitis for intestinal obstruction.

Jaundice, although slight, frequently develops and is dependent upon the coincident obstruction of both the common bile duct and the pancreatic duct (Wirsung) by calculi or by swelling of the duodenal opening.

Suppurative pancreatitis is believed always to be due to infection—primarily, if the ducts become infected from the intestine; secondarily, if the suppuration is due to extension from a neighboring viscus, as a stomach ulcer, and metastatic, by means of the blood current.

The early symptoms closely resemble the hemorrhagic form with which, in fact, suppuration may be associated from infection.

Fever is always present—irregular in course and frequently associated with chills and rigors. The epigastrium is distended and tympanitic, and palpation may detect distinct tenderness over the pancreas or a sense of resistance. The actual abscess can rarely be felt in the early stages. It should be remembered that suppuration may begin in the tail of the pancreas and cause symptoms entirely confined to the upper left side of the abdomen and simulating splenic abscess. Jaundice, according to Fitz, is present in nearly one-fourth of the cases and is due to compression of the common duct by the enlarged and swollen pancreas, to the abscess itself, or even to a stone in the common duct—the starting-point of the whole process. Pus and blood have been noted in the stools from rupture of the abscess into the duodenum.

Usually an abscess develops in the lesser peritoneum and may appear as an epigastric tumor with the other symptoms of a pus collection. By an x-ray examination, the mass is seen to be separate from the liver and spleen. Gangrene and necrosis of the pancreas present but few symptoms and can hardly be diagnosed, unless portions of the sequestrated pancreas are passed with the stools or found at operation.

Differential Diagnosis. — For the purpose of differential diagnosis the acute forms of disease of the pancreas can be considered collectively as one group, remembering that the symptoms are modified by suppuration in a manner identical with other surgical diseases. The symptoms are often obscure, and, as has been said, are mainly those of acute peritonitis originating in the epigastrium.

Intestinal obstruction is the diagnosis usually made, and in fact the two may co-exist, as Robson points out, from constriction of the duodenum by inflammatory adhesions or by the swollen pancreas itself. The obstruction in pancreatitis is at a more unusual site and is not so absolute; and repeated enemas, before peritonitis has supervened, finally restore the peristalsis of the bowel. In favor of pancreatitis are the early fever, rapid pulse, dyspnea, tenderness, epigastric distention, etc., and the absence of general tympany or stercoral vomit. Palpation of the abdominal walls in intestinal obstruction excites storms of pain.

In perforating gastric or duodenal ulcer the tenderness to pressure is less acute and there is less muscular rigidity in the early stages. The history of the patient may suggest gastric ulcer—pain on taking food; the occasional vomiting of blood, if the stomach or the first part of the duodenum be the seat of ulcer; black stools, if the ulcer be in the lower portion of the duodenum; and the chlorotic tendency. It is true that there is often collapse at the time of perforation, but as a rule the immediate shock is followed by reaction, vomiting ceases, tympany becomes general, the lower border of liver dulness raised, and the signs of general peritonitis become manifested.

In cholecystitis the pain, while acute, is of a somewhat different type, is never as severe, and is located over the gall-bladder and radiates to the right shoulder, breast, or back; while in pancreatitis, if the pain radiates it is toward the left. There is a history of intense fever, gall-stone attacks, or gastro-intestinal catarrh, in cholelithiasis as well as in pancreatitis; but in cholelithiasis rigidity is less general and early distention is seldom present.

In acute ptomain poisoning the history of the case, chemical examination of the stomach contents, the more general nature of the colic, and the constant diarrhea should decide the diagnosis.

The following cases of acute pancreatitis have been observed during the past year. Cases I and II have previously been reported,[1] but are interesting enough to bear repetition in this paper:

Case I.—Mrs. M. C., aged 50 years. No family or previous personal history was obtained. Patient was admitted to the hospital in a state of collapse, with subnormal temperature, rapid and feeble pulse, cold, clammy skin, and cyanosis. The abdomen was distended, rigid and tender throughout, with very intense colicky pain in the epigastrium. Death took place 6 hours after admission. From her relatives it was elicited that, 24 hours previous to admission, she had been suddenly seized with violent epigastric pain, nausea, vomiting, and diarrhea. There was no alcoholic history. The woman was quite stout. Post-mortem revealed a diffuse fat necrosis of the omentum and mesentery, which in some places had broken down and undergone calcareous degeneration. The pancreas was enlarged and hard, with a hemorrhagic appearance on section. No gall-stones were present.

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[1] Deaver, Am. Jour. of Med. Sc., February, 1903.

CASE II.—Mr. T., aged 52 years. Family history negative. Had the ordinary diseases of childhood, malaria, typhoid fever (26 years ago), and rheumatism. Uses alcohol and tobacco moderately. Bowels irregular and digestion fair, with some tendency toward dyspepsia. Twenty-five years ago he had an attack of colicky pain in the epigastrium, with vomiting, but no jaundice. The attack lasted 24 hours, and the pain during that time was very severe. Has had 4 similar attacks. Three days before admission he had a slight colic in the epigastrium, vomiting, dyspeptic symptoms (belching and nausea), and noticed that his abdomen was distended and the stools clay colored. On the day before admission he had a sharper attack of vomiting of clear, green, bile-stained material.

On admission the patient was a heavy, stout man, with yellow scleræ, coated tongue, muddy skin, and a distended and tympanitic abdomen. This distention was most marked in the epigastric region, where considerable tenderness could be elicited on palpation. He complained of pain across the lower thorax, which was accentuated by coughing, during which time acute pain was felt over the abdomen. The urine showed the presence of albumin, casts, and bile, but no sugar. Blood examination revealed an anemia and 8200 leukocytes. The stools contained free fat. An x-ray examination showed a shadow, dense in character, over the region of the pancreas.

The patient was kept at rest for a week after admission without any change in his symptoms being observed. The temperature remained between 100° and 101° F., the pulse about 90, and respiration at 28.

On the day before operation there were 13,850 leukocytes (81.3

per cent. of the polynuclear type, and 18.7 per cent. of the mononuclear type). No eosinophiles were observed. Vomiting ceased under treatment, but the strength of the patient was rapidly failing, and operation was decided upon. No mass could be felt at any time in the region of the pancreas, but there was a sense of resistance over the gall-bladder and pancreatic regions, with marked tenderness.

Operation was performed under ether anesthesia, which was administered with difficulty, owing to the cyanotic condition of the patient. An incision was made through the left rectus muscle. The omentum was observed to be speckled with areas of fat necrosis, and the epigastrium was the seat of a peritonitis. The gastrocolic omentum was opened with the fingers and the lesser peritoneum found to be full of a chocolate-like material. This was mopped out with sponges, and rubber and gauze drainage was passed down to the bottom of the cavity. The patient's condition forbade any further interference, and he was sent back to bed. Death occurred 3 hours later.

As no autopsy was permitted the operative wound was merely opened and slightly enlarged. Diffuse fat necrosis was found in the omentum, mesentery, and peritoneal fat. The meso-appendix and the epiploic appendages were involved. There were many areas of calcareous infiltration, some of which had broken down. On widely opening the gastrocolic and gastrohepatic omentums and raising the stomach, the pancreas was found to be necrotic, black, and gangrenous throughout its entirety, with the exception of the head, which, while apparently diseased, was not gangrenous. The entire fat of the lesser peritoneum and adjacent structures, including the perirenal fat, was in a state of necrosis and mostly liquefaction necrosis. The spleen was slightly enlarged. The gall-bladder was distended and filled with gall-stones, many of which were small, and the cystic duct was occluded by a stone. The liver appeared normal. The common duct was free from a stone as far as could be determined, though its course in relation with the pancreas could not be found. The ampulla of Vater was clear. The pancreatic duct could not be made out. No further post-mortem examination could be made. Bacteriologic examinations of the contents of the lesser peritoneum failed to show any growth whatever on blood serum, bouillon, or agar.

Case III.—S., aged 57 years. Married and a gas-fitter by occupation. His mother died of paralysis, his father of obstruction of the bowels. Two brothers and 1 sister are living. One sister died during confinement; 2 children are living, 1 died from enteric fever. No phthisis or malignancy in the family.

The patient had the ordinary diseases of childhood, and rheumatism. Has always been in good health. His bowels are constipated, as a rule, but he is never troubled with indigestion. He is usually a hearty eater and inclined to "biliousness" at times, but he has never had any attack of distinct jaundice or colic. Has been a hard worker.

Three weeks before admission he had influenza. He was sick 1 week, then worked 3 days in the cold, and was attacked with pain in the abdomen, and chills. His bowels were constipated; relieved by oil and followed by obstinate diarrhea. Vomited twice. Had fever, sweats, and chills, and was thought to have tertian malaria. Three days before admission his bowels again became checked and unable to move. His abdomen became swollen and he complained of severe pains in the epigastrium and both hypochondriac regions.

On admission, February 23, 1903, the patient was in collapse, with a weak, irregular pulse and difficult respiration. Pain and tenderness in the upper abdomen, with marked distention. Dulness was noted in the left flank, but no mass could be felt in the epigastrium. No jaundice.

The x-ray showed a dense shadow on the right side immediately below the diaphragm.

The urine contained albumin and casts, but no sugar. The blood counts were: February 23, hemoglobin 87 per cent.; leukocytes 12,240; February 27, hemoglobin 80 per cent., erythrocytes 3,960,000, leukocytes 14,800. On March 1, a differential leukocyte count gave 85 per cent. of polynuclears, 7 per cent. of small lymphocytes, 6 per cent. of large lymphocytes, 1 per cent. of transitional, and 1 per cent. of eosinophiles.

By rest, low diet, stimulation with strychnin and eserin, and ice locally, marked improvement was noted at the end of 24 hours and an operation decided upon, but a few hours later he again collapsed. His condition continued to be very poor for 1 week, with marked exhaustion and delirium toward the end. Death occurred March 3, 1903. The autopsy was performed at the patient's home by the attending physician, who found acute hemorrhagic pancreatitis, acute cholecystitis with a gall-bladder about 4 inches long filled with decomposed bile, but no stones. The head of the pancreas was the site of a cyst the size of an orange.

CHRONIC PANCREATITIS

Etiology.—Chronic pancreatitis may follow the acute form or arise from certain inflammatory processes within the gland. Opie[1] distinguishes two forms of chronic interstitial inflammation: (*a*) Interlobular pancreatitis, with proliferation of fibrous tissue between the lobules, which are invaded from the periphery, and with implication of the islands of Langerhans only when the sclerotic process is far advanced. This form follows obstruction of the ducts, which dilate and dam back the secretion. The effect of this retained secretion seems to be the destruction of the cells of the parenchyma with replacement by connective tissue. The islands of Langerhans are spared because they do not form a communication with the lumen of the ducts. (*b*) Interacinar pancreatitis, much less common, and having a diffused fibrous network invading the lobules, separating the individual acini and invading the islands of Langerhans.

Carnot[2] distinguishes (*a*) mechanical, (*b*) toxic, and (*c*) infectious forms of pancreatitis.

(*a*) *Mechanical pancreatitis* is due to obstruction of the ducts by biliary or pancreatic calculi or by malignant disease of the pancreas or surrounding organs.

A gall-stone in the ampulla of Vater which is large enough to obstruct the common duct will also occlude the pancreatic duct, and the same result may occur from the pressure exerted by a stone in the former somewhat above its junction with the latter. Even with a patent accessory pancreatic duct the entire pancreatic secretion is not discharged, and its retention is followed by alterations in the gland cells and an induration of the interstitial tissue. In addition to this effect of obstruction, the contiguity of the pancreatic ducts to the intestine renders easy their invasion by bacteria and an inflammatory reaction exactly similar to what often occurs in biliary calculi.

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[1] Jour. Exp. Med., 1901, vol. v, p. 397.

[2] Thèse de Paris, 1898.

Pancreatic calculi, usually in the duct of Wirsung, may induce similar lesions and are probably due to some alterations in the chemical composition of the pancreatic juice together with stagnation of the outflow. Perhaps an ascending infectious inflammation of the ducts may cause both conditions.

In a case recently observed by one of the writers pancreatic calculi, completely obstructing the pancreatic duct with a patent, though small accessory duct, were found in a patient who had died of pulmonary tuberculosis. The pancreatic cells were completely destroyed, being replaced by fat and connective tissue. No marked symptoms referable to the pancreas were complained of. The case will be fully reported subsequently.

Carcinoma, compressing the pancreas, may give rise to chronic induration, but the symptoms are referable to the primary disease and need not be discussed.

(*b*) *Chronic pancreatitis due to toxic substances* may result from the misuse of alcohol, and from arteriosclerosis, endarteritis, syphilis, or general tuberculosis.

The relationship between indurative pancreatitis, cirrhosis of the liver, and interstitial nephritis needs to be carefully observed and studied. Opie[1] concludes that "chronic interstitial pancreatitis is not infrequently dependent upon the same etiologic factors, notably alcohol, which produce cirrhosis of the liver, and in about one-fourth of the cases the two lesions are associated." Pancreatitis has usually been considered by clinicians to be a disease of the middle period of life in high livers, and more especially when a free use of alcohol is indulged in, and in those who have a tendency toward corpulency. In our experience, as noted in the cases reported, these facts have not been sustained.

Changes in the walls of the blood-vessels are observed in the pancreas as well as in other parts of the body, lead to an overgrowth of connective tissue, and may produce secondary changes, such as atrophy or fatty degeneration of more or less of the gland, and even hemorrhage and necrosis.

Hoppe-Seyler[2] compares this atrophy to that found in atrophied kidney, the pancreas sometimes becoming a mass of intracinous fat.

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[1] Am. Jour. of Med. Sc., 1902, vol. cxxiii, p. 864.

[2] Deutsche Arch. für klin. Med., 1893, vol. lii, p. 171.

Syphilis produces not only gumma of the pancreas but chronic induration as well, especially when congenital. Schlesinger and Opie have found that the islands of Langerhans are not involved by the new growth of connective tissue nor implicated in the atrophy which affects the cells of the acini. Opie suggests that the disease, like many syphilitic lesions, is one of the interstitial tissue and the changes in the parenchyma result not so much from its destruction as from an interference with its growth.

Tuberculosis, either general or local, is sometimes accompanied by chronic interstitial pancreatitis, and is of interest from its frequent association with diabetes. The symptomatology is not clear, and of course the diagnosis can hardly be made.

(*c*) *Chronic Pancreatitis due to Infections.*—These are usually secondary to an obstruction of the ducts, especially by gall-stones and carcinoma, with an ascending infection from the duodenum; but there may be a chronic pancreatitis without any duct obstruction whatever.

Körte produced chronic inflammation of the pancreas by injecting Bacterium coli into the duct, while Carnot fixed a thread into the duct and extended it through the ampulla into the duodenum, with the production of marked sclerosis of the gland.

Opie has observed four cases in which the clinical history afforded evidence of continued gastric or gastro-intestinal disease, namely, epigastric pain, nausea, and vomiting, the latter repeated and severe. They were presumably the consequence of an ascending infection of the duct.

SYMPTOMATOLOGY.—It is evident that the recognition of this disease is difficult, as nearly all the cases have been reported from the post-mortem room. It is only comparatively recently that surgeons have observed induration of the head of the pancreas when operating upon cases giving symptoms suggesting gall-stone or malignant obstruction of the common duct. One of the writers has frequently recorded such cases on the records of the German Hospital.

This can be easily understood by the brief description given of the etiology of these chronic lesions of the pancreas. Long before the disease of the pancreas can give rise to symptoms, the gastro-intestinal disorders, gall-stones, arteriosclerosis, etc., will have shown some manifestations of their presence, and the later symptoms, in nearly the same locality, will be supposed to be a continuation of the primary lesion. Furthermore, it is probable that disturbances in the functions of the stomach, intestines, and liver must take place when the causes mentioned induce a lesion of the pancreas—so that the symptomatology observed is very complex. The clinical laboratory does not give us any aid at present in the way of tests to determine the effect of the retention or perversion of the pancreatic secretion. That this is so is due to the adaptability of the organism whereby the fat may be split by a ferment in the stomach or decomposed by micro-organisms lower down in the intestine, as well as by the normal action of the bile.

Pancreatic diabetes is coincident with disease of the islands of Langerhans, and as these are not involved in the interlobular form of pancreatitis due to obstruction of the duct, glycosuria is absent.

When pancreatitis is due to acute obstruction of the pancreatic duct or to infection from the intestinal canal, the onset is usually characterized by sudden, intense pain in the epigastrium, with nausea and vomiting, the latter frequently very severe. The attack is mistaken for gall-stone colic in the great majority of cases, more especially as jaundice is frequent from an infection or obstruction of both ducts. During the attack the recti muscles become rigid and a point of tenderness may be detected over the head of the pancreas, 1 inch above and 1 inch to the right of the umbilicus. In a few cases the stools may appear greasy and lighter in color, and lipuria and biliuria may be found.

Glycosuria, especially in an alcoholic subject, with a cirrhotic liver is a valuable sign.

As the disease advances gastro-intestinal disorders become constant, with emaciation, loss of strength, and recurring attacks of epigastric pain and jaundice.

In thin subjects a tumor may be made out, and if malignant disease can be excluded the diagnosis becomes clear; but such a differentiation is extremely difficult, the indurated pancreas frequently producing a permanent jaundice, with enlarged gall-bladder. In advanced stages with destruction of the parenchyma of the gland, glycosuria is more common, and the patient finally dies of exhaustion, or perhaps some terminal infection.

In cases associated with syphilis, arteriosclerosis, or tuberculosis, the onset is probably much slower, more general, and indefinite. Sensations of pressure in the epigastrium, gastro-intestinal disturbances, epigastric tenderness, and emaciation may be complained of.

Some patients undoubtedly recover after having lost much weight, and with a history of gall-stone colic, jaundice, and anemia. The duct of the pancreas having again become patulous, the secretion is no longer retained, and sufficient healthy parenchyma remains to perform the pancreatic functions.

Azotorrhea.—While the digestion of albumin is not performed entirely by the pancreas, its disturbance, according to Oser, is a sign of considerable value, if, in the absence of jaundice or gastro-intestinal disease, there are also present disturbances of the cleavage of fat; while azotorrhea is a certain sign of pancreatic disease if a tumor can be felt in the region of the pancreas.

A test of deficient pancreatic secretion often given is the administration of salol, which is split up by the pancreatic juice into salicylic acid and phenol. The latter is excreted in the urine and if absent after the administration of 5 grams of salol in divided doses in 24 hours, the pancreatic secretion is supposed to be wanting.

Moynihan [1] made a correct diagnosis of chronic pancreatitis with calculus upon the following symptoms: "Steady loss of weight; gradual wasting; irregular pigmentation of the skin, in patches, of the color of café-au-lait (very closely resembling the pigmentation of Molluscum fibrosum); persistent attacks of epigastric pain and uneasiness of the type of hepatic colic, though less severe and unattended, until very late in the history, by jaundice, which was then always trivial, though unmistakable; and pain passing through from the front of the abdomen to the middle of the back. There was no rigor nor any complaint of sensations of heat or cold. The stools were occasionally 'frothy' and 'greasy.' On examination under chloroform some indefinite swelling could be felt about the umbilicus and a little to both sides of the median line, though chiefly to the right."

DIFFERENTIAL DIAGNOSIS.—The differential diagnosis must be made from gall-stones in the common duct, carcinoma of the pancreas and bile ducts, and perhaps chronic gastritis or perigastric adhesions with pyloric stenosis.

While the diagnosis of gall-stones is not so important, both conditions requiring operation, yet the knowledge that pancreatitis exists is a great aid to successful operative treatment.

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[1] Lancet, August 9, 1902.

In gall-stone obstruction the pain is more often referred to the back and shoulder, the liver and gall-bladder are tender to pressure and frequently enlarged, and there is usually a history of repeated attacks with intermittent jaundice and periods of complete repose. The pain of gall-stone obstruction in the common duct is sometimes differentiated from that in pancreatitis by being located to the right of the middle line. We have not found this to be so, many cholelithiasis cases having well-marked pain in either the epigastrium or the left side, especially with a low position of the stone and without obstruction of the pancreatic ducts.

From carcinoma of the pancreas the diagnosis is difficult and seldom made. The more marked cachexia and secondary anemia, absence of pain or tenderness, painless enlargement of the gall-bladder, extreme jaundice, ascites, and the enlarged, smooth liver, are the symptoms observed most constantly in malignant disease. When the liver is involved, nodular masses may be palpated.

In carcinoma of the bile ducts the gall-bladder is much dilated. Jaundice is absent unless the portal lymphatics become enlarged and cause pressure, or the infiltration of the walls occlude the lumen. Later cachexia, secondary anemia, and sometimes ascites develop.

In malignant disease of both the pancreas and the biliary tract careful questioning of the patient will determine the nature of the onset.

Chronic induration of the pancreas, as has been frequently mentioned, is due in the great majority of instances to obstruction or infection of the ducts, and the class of cases beginning with sudden epigastric pain and vomiting will be found greatly in the majority.

In like manner chronic gastritis, gastric ulcer, and pyloric carcinoma can be separated from the pancreatic lesion by careful attention to the minute points in the previous history of the patient. The stools should always be searched for fat and muscle-fibers, the urine for sugar, phenol (salol test), and the fat-splitting ferment. If gastric complications are suspected the test meal is invaluable and the blood count will show the qualitative changes in the red and white cells. The clinician, by careful palpation and percussion, can ascertain the point of greatest tenderness and resistance with the possible finding of a tumor mass. An x-ray examination may show a mass in the region of the head of the pancreas, *moving with respiration.* Such a finding is very valuable, as the shadow cast by the pancreas is not readily movable with the motion of the diaphragm. (Wilbert.) It is only by grouping both the clinical and the laboratory findings that disease of the pancreas can be accurately and satisfactorily studied.

The following six cases of chronic pancreatitis were so diagnosed before operation, with the exception of Case VI, which was thought to be malignant.[1]

Case IV.—E. O., aged 44 years. Family history is negative. During early life he had measles, whooping-cough, scarlet fever, varicella, mumps, and diphtheria; but since 12 years of age he enjoyed almost perfect health, until 1890, when he had an attack of diarrhea lasting for nearly 4 years and more or less severe in character. Appetite is always good, and no dyspeptic symptoms have been experienced. He smokes 5 or 6 cigars daily, uses alcohol moderately, and is a man of very good habits.

In 1891 he had a sudden and severe attack of pain in the epigastrium, lasting 2 or 3 hours, then passing downward through the abdomen, and followed by diarrhea; no vomiting. The pain was colicky in nature, and usually relieved by sinapisms. A number of attacks recurred at intervals of 6 months. In July, 1901, he had an attack similar to the preceding, but more severe in character. It lasted about 2 hours, and was relieved by the application of a strong red-pepper plaster; no vomiting. After this attack he enjoyed exceptionally good health until June, 1902. Owing to overwork, he became depressed and nervous. His physician found that his heart action was weak and irregular, and prescribed rest. Edema of the feet was present, and a urine examination excluded nephritis. He then became progressively weaker, with anorexia, and again suffered from diarrhea and pain in the region of the epigastrium, colicky in nature but nearly constant. There was a sensation of bloating present. During the week previous to admission there was constant epigastric pain, with marked constipation, and with rectal tenesmus and desire (without result) to have a bowel movement. No nausea or vomiting; no urinary symptoms.

On admission the patient was flabby and rather stout, with a pale skin of a dull ashen color. The scleræ were yellow, the tongue coated, and the breath foul. The pulse was rapid, and the patient markedly nervous. The heart was weak, with a tendency to irregularity. The lungs were clear, and no apparent enlargement of the liver, gall-bladder, or spleen could be noted. There was no albumin, sugar, or biliary coloring matter in the urine. The epigastrium was tender and the abdomen distended. No palpable tumor could be detected over the pancreas, but chronic pancreatitis was diagnosed.

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[1] Cases IV, V, and VI have previously been reported (Deaver, loc. cit.).

Operation under ether anesthesia revealed a hardened pancreas, enlarged at its head, without fat necrosis. The bowels were carefully gone over from the sigmoid to the pylorus, and no constricting bands were found, nor was there any peritonitis beyond the early stages of hyperemia. The gall-bladder was distended and somewhat enlarged, while the liver was light in color and probably fatty.

A cholecystostomy was performed, with the difference that the gall-bladder was sutured to the peritoneum and aponeurosis without opening it, and gauze drains were introduced not only around the gall-bladder, but to the space below the liver.

The patient reacted very well from the operation, and 2 days later the gall-bladder was opened with the actual cautery, and rubber drainage introduced into the gall-bladder. Seven days after operation part of the gauze drainage was removed, and 13 days after operation the remaining gauze was taken out. The patient then became rapidly convalescent.

Case V.—S. Z., aged 55 years. One brother died of tubercular enteritis. The patient has suffered from measles, whooping-cough, influenza, and malaria. His bowels are irregular and appetite good. He is a moderate user of alcohol. The attack of malaria occurred 20 years ago, and was accompanied by jaundice, lasting for 3 or 4 weeks, and colicky pain in the epigastrium, followed by irregularity of the bowels and dyspeptic symptoms for some years. Six weeks before admission he was seized with severe pain in the epigastrium, colicky in nature, and referred to the umbilicus; 1 week later he became jaundiced, with intense itching. The urine was dark, and the stools became lighter. No vomiting.

On admission the patient was markedly jaundiced, with a slow pulse. He was a large man, with considerable adipose tissue. Examination of abdomen revealed the liver enlarged, but not tender. The gall-bladder was large, palpable, and very tender, and an x-ray revealed a dense shadow in the epigastrium to the right of the median line. The urine contained albumin, casts, and biliary coloring matters, but no sugar. The blood showed a marked anemia, 5000 leukocytes, and delayed coagulation. There were no gall-stones in the stools.

Operation was performed under ether anesthesia, 8 days after admission. The gall-bladder was much distended, fluctuating, and non-adherent; it was aspirated of 350 c.c. of thick bile, and the opening closed with a hemostat. No gall-stones were detected in the gall-bladder, cystic, or common ducts. The pancreas was found to be enlarged, hard, and infiltrated. A cholecystoduodenostomy was performed, using a small Murphy button. The wound was closed with silk tier sutures, and except for a small stitch abscess the patient made an uninterrupted recovery and was discharged 28 days after operation. His jaundice had nearly faded, and the stools were of a natural color.

Case VI.—J. C. K., aged 58 years; a shoemaker by occupation. The family and previous personal history was negative. He used alcohol moderately. His position while at work caused occasional pains in the epigastrium, but never vomiting. He had never, to his knowledge, passed blood or gall-stones in the stools. Six months before admission he suffered from weakness, anorexia, and coldness of the extremities, and was placed on a milk diet, which caused marked constipation. Three weeks later he became jaundiced, which deepened, but without pain other than an occasional colicky attack to the right of the epigastrium. He lost weight—23 pounds—during the 6 months.

On admission the heart and lungs were normal, the spleen not enlarged. The temperature and pulse normal. He was profoundly jaundiced and emaciated. The urine contained a trace of albumin, no casts or sugar, a normal amount of urea, absence of indican, and presence of bile. A stomach analysis showed a trace of hydrochloric acid, the presence of lactic acid, and a total acidity of 12. The stools were negative for free fat or gall-stones. A number of blood examinations revealed an anemia, no leukocytosis, and delayed coagulation. The latter was not benefited by the use of calcium chlorid for the 4 days preceding operation. An examination of the abdomen found the recti muscles rigid, especially in their upper quadrants. To percussion an area of dulness could be detected over the region of the pancreas and extending above and to the right and merging with the liver dulness. The inguinal lymphatics were enlarged. A fluoroscopic examination revealed a diffuse shadow around the head of the pancreas.

Operation was performed 4 days after admission, under ether anesthesia. The gall-bladder was found to be small and adherent to the liver, with a large stone in the cystic duct. A cholecystectomy was done and a peritoneal cuff sewed over the stump. Following the common duct with the finger in the foramen of Winslow, a round, hard mass could be made out, presumably a stone in the common duct near the duodenum. The abdomen was deep and examination difficult, but the common duct was dilated to 3 times its normal size. The duodenum was opened opposite the ampulla, and the obstruction was found to be the head of the pancreas; the duodenum was therefore closed and opened behind, and a choledochoduodenostomy performed, with silk suture. An incision was then made in the right loin and gauze disposed about the anastomosis, leading across the stump of the gall-bladder and thence under the liver and out the loin. A rubber tube and two wicks of gauze were used to drain from the anastomosis through the abdominal wound. The abdomen was partly closed. The patient left the table in fairly good condition, with pulse of 72. There was a continuous capillary oozing, however, and the patient grew weaker, the pulse feebler, and death took place 24 hours after operation.

A post-mortem revealed a chronic pancreatitis, mitral insufficiency, nutmeg liver, and chronic congestion of the spleen and kidneys. There was no fat necrosis.

Case VII.—Rose A., aged 48 years, married; Italian; occupation, housewife. Her father died of cholera, her mother of old age. Two brothers are living. One brother died of an accident and 1 sister of pneumonia. No phthisis nor malignancy in the family. The patient had ordinary diseases of childhood, and enteric fever at 22. She has had 8 children and 1 miscarriage. Menstruated at 11; menopause at 47½. Always well, bowels regular, appetite good. Fifteen years ago began having sharp pains in right hypochondrium every month. Never jaundiced. Lasted a few hours and then disappeared. Umbilical hernia for 15 years, but no trouble.

Three weeks before admission she had severe cramps in right hypochondrium, lasting 1 day; never vomited. Eight days before admission she had another attack of colic in same locality. Bowels regular, no vomiting.

On admission the abdomen was tender in the epigastrium and over the gall-bladder; slight jaundice. After admission she had another severe attack of colic lasting several hours. The urine was amber, acid, and had a specific gravity of 1024; trace of albumin, hyaline casts, and bile; no sugar at any time. The leukocytes were 11,800.

Operation on November 26, under ether. Incision was made in the median line to ascertain the condition of the hernial sac. The omentum was adherent and was ligated and excised. An exploration of the abdomen showed the appendix and pelvis normal, the pancreas swollen and indurated, and the gall-bladder distended. The hernia was repaired and a second incision made over the gall-bladder, which was sewed to the peritoneum and fascia. Two days after operation the patient was in good condition and the gall-bladder was opened with the cautery. Thick, muddy bile escaped, and 80 small stones were removed. During the following 2 weeks 144 additional stones were removed, and on discharge, January 6, 1903, recovery was complete.

Case VIII.—Mary W., married, 29 years old, and a housewife. Her father died of "dropsy," her mother of Bright's disease. One sister is living and well, 1 dead of heart disease. Four children are living and well, youngest 5 months old. No miscarriages nor any history of malignancy or phthisis. She had ordinary diseases of childhood. Menstruated first at 13, duration 3 days, regular, and with dysmenorrhea. Constipated, the stools usually being dark in color. No urinary trouble. Slight leucorrhea.

About December 15, 1902, she was suddenly seized with cramp-like pains in the upper abdomen referred to the back and right shoulder. The region of the gall-bladder and pancreas were markedly tender and nausea and vomiting of bile concomitant. The attack subsided, but was followed by jaundice which soon faded. Pain continued over the head of the pancreas until February 13, 1902, when she was again seized suddenly with colic, nausea, vomiting, and anorexia. The taking of food increased the pain. No hematemesis; some headache.

On admission, February 17, 1903, she complained of pain over the region of pancreas and gall-bladder radiating to the back. Also pain over the same region upon coughing or deep respiration, tenderness, and rigidity over the right rectus. The gall-bladder was not palpable, the liver not enlarged, and the lungs and heart normal. Stomach capacity 1675 c.c., with the greater curvature one-half inch above the umbilicus. Vaginal examination reveals nothing pathologic. Urine dark amber, acid, 1028, trace of albumin, no sugar; contains hyaline and granular casts. On February 19 salol was administered, and carbolic acid subsequently examined for in the urine and found. On February 18, the hemoglobin was 83 per cent., the leukocytes 7040. On February 18 test meal was administered and analysis gave: Total acidity 7 per cent., no lactic nor free hydrochloric acids present. Microscopically some blood and bile pigments were observed.

February 19. Coagulation time of blood, 3 minutes.

February 20. Stool examined showed presence of incomplete albumin digestion; no free fat nor calculi.

February 20. X-ray examination gave a large but not very dense shadow to the right of the median line, the lower portion of which appeared to be fixed and allowed the upper portion to slide down over it. The patient had quite a wide range of diaphragmatic movement.

A diagnosis was made of chronic pancreatitis, cholelithiasis, and dilated stomach.

Operation was performed February 21, under ether anesthesia. The head of pancreas was found to be enlarged to the size of a fist and hard. The gall-bladder was opened and several calculi, mulberry in appearance, were removed. The common and hepatic ducts were explored and found to be clear. No stones were found in ampulla of Vater. No fat necrosis.

Cholecystostomy was done. The patient recovered and was discharged March 16.

Case IX.—C. B., aged 60 years, married. Father died from an accident, mother of phthisis. Two sisters and 2 children are living. No other history of phthisis nor malignancy in the family.

One year ago the patient had a sudden attack of colic in the epigastric region, with backache. The attack of colic lasted 2 days, was severe, and prevented work for 2 weeks; vomited excessively. There was much jaundice lasting 1 week. Constipation. Color of feces not noted. Full recovery. Was never a heavy drinker. Good health until 3 hours before admission, when he was seized with severe chill, epigastric pain, and vomited green fluid. Bowels were open but the color was not noted.

On admission, November 25, 1902, the patient was cold, and in collapse. There was epigastric tenderness and rigidity. The gall-bladder was tender. Scleræ were muddy, the tongue clean. Belched considerable gas. An indistinct mass or muscular rigidity over the gall-bladder was present. Right-sided inguinal hernia.

Urine was dark amber, had a specific gravity of 1036, and contained albumin, hyaline and granular casts; no sugar at any time. Blood count: Hemoglobin 75 per cent., erythrocytes 4,330,000, leukocytes 11,700. The stools contained no stones. X-ray showed large, dense, and rather sharply outlined shadow on the right side below the diaphragm. A diagnosis was made of chronic pancreatitis and cholelithiasis.

An operation was done November 26, 1902, under ether anesthesia. The pancreas was enlarged and nodular and the gall-bladder the seat of old inflammation, and adherent. The gall-bladder was much distended. Stones were in the common duct, which were easily displaced into the gall-bladder. Cholecystostomy performed as usual.

The patient reacted well from the operation and the fistula continued to discharge stones until December 6. On discharge from the hospital, January 5, 1903, there remained a small biliary fistula which did not close until February 7, 1903. One week later he was suddenly seized with severe paroxysmal pain in the epigastrium and right hypochondrium, with severe vomiting and rapid onset of jaundice. A large tender mass was noted over the gall-bladder, which was opened and 1000 c.c. of thick, yellow fluid escaped. The pain was relieved, but the patient was sent back to the hospital.

On admission, February 17, his condition was poor with a leaky skin, coated tongue, slight jaundice, and rapid weak pulse. Some tumefaction was found over the gall-bladder and the wound appeared clean, but was evidently not draining. The urine was bile stained and the stools clay colored.

The second operation was performed on February 20, under ether anesthesia, and the gall-bladder region was found to be the site of numerous adhesions, the organ itself having closed and containing 200 c.c. of thick, yellow fluid. Drainage was again introduced into the gall-bladder. *The pancreas was found to be just one-half the size observed at the previous operation.*

Despite this second operation the fistula persisted, practically no bile reaching the intestine, although the patient made a full recovery. It was evident that some obstruction still existed in the patency of the common duct, and to correct such a condition a third operation was attempted on April 15, 1903.

The adhesions encountered about the gall-bladder, duct, stomach, and intestine were of such a firm nature that further manipulation was deemed hazardous, *especially as the pancreas was found to be perfectly normal in size.* A cholecyst-colostomy was accordingly performed, using a Murphy button to perfect the anastomosis.

From this operation the patient recovered perfectly and is in good condition at the present time.

CYSTS OF THE PANCREAS

Cysts of the pancreas, while comparatively rare, have been observed and operated upon a number of times and the diagnosis can be more readily made than in the inflammatory affections.

The cysts are more frequently present from the twentieth to the fortieth year, and are about equally common in both sexes.

Körte divides them into: (1) Traumatic, following blows or continued pressure upon the abdomen; (2) post-inflammatory; and (3) cysts without any apparent exciting cause. In this class of cases the cysts existed for a very long time and caused but few symptoms.

Mayo Robson gives this anatomic classification: (1) Retention cysts; (2) proliferation cysts, cystic adenoma or epithelioma; (3) hydatid cysts; (4) congenital cystic disease; (5) hemorrhagic cysts; (6) pseudocysts.

Retention cysts are common in the salivary glands, which are closely related in structure to the pancreas. Retention of secretion in the pancreas is probably the most frequent cause of these cysts. Chronic induration, calculi or malignant disease of the pancreas, duodenal tumors, adhesions, swollen lymph glands, or stones in the bile duct may cause such compression of the main or smaller ducts as to induce dilatation of them and of the alveoli with the disappearance of intervening walls, and thus by fusion a cyst is formed.

Proliferation cysts are rare and may be considered as true primary cystic degeneration of the pancreas, or as due to cyst formation within tumors.

Hydatid and congenital cystic diseases are very rare conditions.

Hemorrhagic cysts are perhaps always due to the accidental presence of blood, from the tendency toward bleeding which the pancreas exhibits when diseased. Yet it seems reasonably possible that bleeding within an area of the soft pancreatic tissue can be the origin of these cysts.

Pseudocysts are collections of fluid in the lesser peritoneal cavity more or less closely related to the pancreas, usually due to injury. They may or may not contain traces of the pancreatic ferment, depending upon the injury done to the pancreas.

The situation of the pancreatic cysts is variable. Any part of the gland may give rise to them, but the tail is the most frequent site. The direction of the growth is always variable; the greater number developing in the lesser peritoneal sac, push the stomach upward and to the right and emerge between the stomach and transverse colon. In some cases the tumor reaches the abdominal wall above both the stomach and colon, pushing them downward; in other instances the cyst is below the colon and at the lower portion of the lesser sac, between the two layers of the transverse mesocolon. Occasionally the cyst extends in the direction of the spleen or the kidney, and indeed the origin of such a cyst may have been from the kidney or suprarenal body. As a rule the cyst is adherent to the surrounding structures, especially to the stomach, colon, duodenum, omentum, and anterior abdominal wall. Some cases are free from adhesions.

SYMPTOMATOLOGY.—The symptoms of a pancreatic cyst are dependent in great measure upon the pressure exerted upon surrounding organs, but it must be remembered that the majority of such cysts are due to retention of secretion from obstruction, usually chronic pancreatitis. Accordingly the early symptoms may be those of chronic pancreatitis, vague and obscure, until the cyst can be actually palpated. The stagnation of the secretion and the interference with the function of the gland will react upon the system with disturbances of nutrition and dyspeptic symptoms.

Pain is the most constant symptom, and may be continuous or colicky in nature. In neither of the cases herein reported was pain a symptom of any moment. Both patients complained of epigastric discomfort at times, but no distinct pain. Vomiting is usually present, is more marked after eating, and is in direct proportion to the amount of pain suffered. Our two cases were free from pain and never complained of vomiting. Constipation is the rule, and jaundice may be present and is dependent upon the obstruction of the common bile duct. Wasting frequently results, with advancing weakness. The presence of sugar in the urine, fat and undigested proteids in the stools, and decolorization of the stools has been observed, but is not constant.

The ureters may be compressed and induce a hydronephrosis, and interference with the circulation of the abdomen may result in ascites or edema, as was observed in one of the cases following.

The presence of a tumor is of course necessary to make the diagnosis. The mass is nearly always found in the left side of the epigastrium or left hypochondrium, because of the greater frequency of cysts in the tail and body of the pancreas. The surface of the cyst is smooth and as a rule feels as if tensely stretched. Fluctuation is usually present, though often indistinct. The tumor may move with respiration or may permit displacement laterally, while percussion shows the dull note of a mass of liquid or tympany from the overlying stomach and colon.

Differential Diagnosis.—The differential diagnosis must be made from renal tumors, such as hydronephrosis, from enlarged spleen, ovarian cysts, hydatid cysts of the liver, mesenteric cysts, retroperitoneal sarcoma, and large abdominal aneurisms. The previous history and slow onset, with progressive weakness; the deep situation of the tumor and its slight range of mobility; the condition of the stools; and, further, the fact that the great majority of cystic tumors in the epigastrium are pancreatic, should aid in the diagnosis.

In hydronephrosis there is usually a history of renal colic, intermittent attacks of polyuria, and often blood and renal epithelium in the urine. Catheterization of the ureters will perhaps decide whether the kidney is performing its function. This procedure may be feasible in the female, but the writers question whether any but the most expert can reach the ureters in the male. This is contrary to oft-published teaching, but is nevertheless a fact. The family physician who will encounter such cases is never able, even if competent, to catheterize the ureters. The mass in the loin can be more readily grasped than in the pancreatic cyst and palpated between the hands.

Ovarian cysts, when small, are easily diagnosed, but when the tumor within the abdomen is very large some difficulty may be encountered. The history of an ovarian cyst should show the origin to be from the pelvis upward, and examination of the pelvis in the Trendelenburg position may enable both ovaries to be readily felt; and by inflation of the stomach with air the ovarian cyst will be found to crowd it backward.

Mesenteric cysts are characterized by great mobility, especially in the transverse direction; they may be rotated upon the axis, and they are frequently surrounded by a resonant zone, as well as crossed by a band of resonance. This is easily understood when it is recalled that the cyst grows between the leaves of the mesentery.

The following cases occurred at the German Hospital during the past three years:[1]

Case X.—Mrs. E. S., aged 58 years. One brother died of tuberculosis. Previous history negative. Menopause at 40 years of age. Her habits were good. Three years ago she had a "typhoid malarial" infection, as she termed it, which lasted for a number of months (evidently was a long and mild attack of enteric fever). A few years after the menopause she became stouter and corpulent, and has continued slowly to increase in girth of the abdomen. Constipation was the rule, and occasionally she would have difficulty in micturition. She was sent to the hospital and the case diagnosed as ovarian cyst.

On admission the patient was a large and stout woman, with a circumference at the umbilicus of 52 inches. She suffers no pain. Examination of the abdomen revealed enlarged and tortuous superficial veins, general fluctuation, and deep-seated tympany in the right flank, and dulness but not flatness in the left flank. The anterior surface of the abdomen was flat to percussion, extending upward to the ensiform, and the area of dulness was not altered by any change of position. No outlines of a cyst or mass could be definitely made out, though there was a hard, cartilage-like body 5 cm. below and 5 cm. to the right of the ensiform, about 4 by 3 cm. in size and just beneath the abdominal wall. The lower border of liver dulness was displaced upward for 5 cm. The apex beat was pushed slightly upward and to the left. Vaginal examination was negative.

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[1] See Deaver, loc. cit.

The urine was free from albumin and sugar, and a blood count showed marked anemia, with a normal leukocyte count.

Under ether anesthesia, an incision 7 cm. in length was made in the median line above the pubes. On opening the peritoneum about 6000 c.c. of ascitic fluid was evacuated. It was then possible to determine that a large cyst was present in the upper abdomen, adherent to the anterior abdominal wall, colon, omentum, and other viscera, and by an adhesive band to the right broad ligament. The incision was continued upward to the ensiform, and the adhesions found so extensive that the removal of the cyst was totally impossible. The lower three-quarters of the wound was sutured, leaving glass and gauze drainage in the pelvis. The cyst was aspirated of 500 c.c. of an oily yellow fluid, its edges sutured to the aponeurosis and peritoneum, and the cavity of the cyst drained with gauze and a rubber tube.

The patient made an uneventful recovery, but the fistula from the cyst was not closed. An examination of the fluid aspirated from it revealed it to be oily, thick, and yellowish in appearance. On standing it separated into 2 layers—the upper yellow and curdy, the lower greenish-brown and thinner. Microscopically, cholesterin crystals and a few epithelial and blood-cells, and an unrecognizable, colloid-like material were found. The specific gravity was 1023. It contained much albumin, but no sugar; was neutral or slightly acid in reaction. No methemoglobin. Cultures on bouillon and agar showed negative results. The fluid digested albumin without the presence of an acid.

Case XI.—Mrs. McC., aged 58 years. The family and previous history was negative. She has had 9 children and 5 miscarriages. Menopause when 50 years of age. Thirty years ago, without any known cause or trauma, she noticed on awakening that she had a mass over the stomach and just below the left costal margin. This was not accompanied by pain or vomiting. It was about the size of a large orange. No jaundice at any time; no diarrhea. The tumor has slowly increased in size and has become more apparent on examination on account of loss of weight in the past 2 months. She has never had any discomfort from the tumor mass, and suffers no dyspeptic symptoms.

On admission the patient was a stout woman without any subjective symptoms. The lungs were clear and the spleen and liver dulness normal. Her abdomen was flaccid, and revealed the presence of a tumor extending from the umbilicus to the diaphragm, moving with respiration, slightly fluctuating, and slightly movable by the examining hand. It occupied the whole epigastric space, and was evidently a cyst in the lesser peritoneum. The urine was negative. Blood count showed the absence of oligocythemia, and a marked eosinophilia. Gastric analysis revealed a trace of lactic acid, a total acidity of 20 per cent., and the absence of free hydrochloric acid.

Operation was urgently advised, but was refused. She is still in good health at the present time, 10 months after discharge from the hospital.

Treatment

TRUNECEK'S SERUM IN ARTERIOSCLEROSIS

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I WISH to propose that Trunecek's name should be given to the solution that he has had the merit of recommending in the treatment of arteriosclerosis.

In his publication he laid special stress on the improvement effected by its use in the subjective symptoms connected with the condition of the large vessels, and on the relief it gives in cardiac asthma. I, in turn, applied this mineral serum to the treatment of cerebral arteriosclerosis, and was then led to test it in sclerotic otitis. In a series of papers that have been published, or that are still in preparation, I have endeavored to fathom the mode of action of concentrated serums, and have searched for new applications of these solutions to therapeutics.

The study of this serum, even when confined to the treatment of arteriosclerosis, is still far from complete; but it is well to decide what results have been obtained, even though many of the publications on the subject are quite recent. It is to our interest to show what we can hope to obtain from this method; but at the same time we must not fear to criticise closely what has been published, and to warn the public against the exaggerated hopes that a new treatment is apt to awake at the beginning. It is advisable to be on one's guard lest the enthusiasm of the beginning be followed by a reaction in the other direction, and lest by overpraising a drug, to begin with, we may cause it to be unjustly abandoned in the end.

Trunecek's serum is composed as follows:

Sodium sulphate	0	44
Sodium chlorid	4	92
Sodium phosphate	0	15
Sodium carbonate	0	21
Potassium sulphate	0	40
Distilled water sufficient to make	100	

It represents, but in a solution ten times more concentrated, a mixture of all the alkaline salts that constitute the inorganic part of the normal blood serum in their proper proportions. The question might be asked how Trunecek came to choose a series of figures that no one could possibly remember? I think that I have found the reason: they are the very reproduction of the figures of Hoppe-Seyler's analysis of blood serum, with only two modifications. According to that chemist blood serum contains 0.73 per 1000 of phosphate of lime and magnesium, while it contains no sulphate of potassium at all. It is, I fancy, owing to theoretical ideas, which I think are mistaken, that Trunecek has suppressed the former elements, while he has added a potassium salt, although potassium does not exist in the serum but in the clot. In a word, his formula is Hoppe-Seyler's, but modified.

A few remarks may now be useful, from a practical point of view. This solution must naturally be made with distilled water, and then sterilized; but this sterilization in the autoclave requires precautions, otherwise there may be precipitation of salts, and the injection may then become painful, or be the point of departure of a small induration. The best plan is to sterilize in a porcelain bougie. When large quantities of serum are needed, as in hospital practice, it is well to add to the solution, prepared with boiled, distilled water, some antiseptic substance, and personally I prefer resorcin, of which I add one gram per liter.

Trunecek injects 1 c.c. of the solution, and repeats the injection every 4 to 7 days, increasing the dose by 0.1 to 0.5 c.c. each time. In some cases he makes an injection every day, and has gone as high as 5 c.c. or even 7 c.c. per dose. He usually makes the injection in the forearm.

I have modified his practice in some particulars. As with all similar injections it seemed to me that the parts of the body rich in cellulo-adipose tissue are to be preferred, and as a general thing, especially with men, I choose the hip. I have thought that it was preferable to make a superficial and not a deep injection. I generally begin with 2 c.c., increasing by 1 c.c. every day, and remaining at 5 c.c., except when there is much pain. I agree with Trunecek in making the injection slowly; furthermore, it is well to insert the needle first, to see whether any blood comes, in which case another puncture should be made, as this occurrence favors the formation of small indurations.

Injections should be made in the right and left sides in turn. In special cases I have injected as much as 12 c.c. in two places, and I think that this dose can be increased still more, even when administered in the form of hypodermic injections; on the other hand, it may be advantageous in certain cases not to resort to large doses, as some patients seem to do better on small ones.

The patients sometimes complain of a feeling of stricture, distention or numbness in the limb in which the injection has been made, but as a general thing the pain does not last. The expedient has been tried of giving an injection of a few centigrams of a 2 per cent. solution of cocain a few minutes before the serum injection; but I do not favor this practice, as the action of the cocain appears to modify the effect of the inorganic serum.

By the side of the injection method can be placed the administration of the serum by enema. Up to the present time I have found that the natural solution is tolerated up to a dose of 35 c.c. without any inconvenience, and further on I will give the comparative results given by the hypodermic and enema methods; but I can already say that I think there is advantage in alternating the two methods. As a general thing I begin with ten injections, after which I continue the treatment by enemas, coming back to the injections if necessary. The action of the serum shows itself very quickly, but it disappears equally quickly; it is therefore well to stop the treatment now and then, and to give the patient a rest. The question whether similar results can be obtained by giving the serum by the mouth is one that I am now examining, but in this regard I have no satisfactory data as yet.

I now pass to the results that are given by this method.

Trunecek's serum is applicable to sclerosis of the large vessels, to sclerosis of the heart, and to arterial cachexia. It is indicated in cerebro-arterial sclerosis, and in chronic rheumatism. These are the first facts that have been demonstrated, and that must be proved in detail.

The first of the four cases mentioned by Trunecek was that of a subclavian aneurism, the size and shape of a chestnut, in a man of 44 years, with atrophy and loss of motion of the right arm, and dyspnea and palpitations rendering the exertion of walking almost impossible. After the iodid treatment had been tried for three weeks without effect, a first injection of 0.3 c.c. of serum was made, and another of 1 c.c. ten days later. In a few hours' time the patient declared that his dyspnea had gone, as well as his disagreeable sensations and palpitations in the region of the heart. The writer says that at the end of seven weeks the pulsation felt over the aneurism had completely disappeared; the dimensions of the tumor became very small after 50 injections, or 190 c.c. in all; and the patient's general condition remained satisfactory, even after the injections had been suspended for three months.

I am fortunately able to report a case similar to Trunecek's, which will place me in a position to criticise the latter in a certain measure. The following is a brief outline of this case:

The patient is a woman, 54 years of age, without hereditary or personal pathologic antecedents, with the exception of an attack of bronchitis five years ago; she is suffering from a double aortic lesion, and furthermore from an aneurism of the right common carotid artery. When I first saw this patient three months ago she had been suffering for 20 months from nocturnal crises of dyspnea, with sensations of torsion in the epigastrium and propagation in the form of painful cramps into both arms. These attacks were of two kinds: some were accompanied by atrocious pain, forcing her to cry out, which led her neighbors at first to think that she was being confined; these attacks lasted two hours or more. The other attacks, which were less long and painful, obliged her to sit up in bed for half an hour. When she came into my hands the severe attacks had become more frequent, and occurred as often as three times a week.

During the preceding 20 months she had been put on a variety of treatments, among others on an absolute milk diet for two months, on a mixture of iodid and bromid, and on valerianate of ammonium; but at no time since the beginning had she passed a night without suffering.

I then began to treat her with Trunecek's serum, starting with an injection of 1 c.c. Guided by her experience of the past she fully expected a very severe attack the same night; whereas for the first time for 20 months she had only a slight attack of pain lasting from 15 to 30 minutes. On the following day the injection was repeated, and her attack of pain was only a slight one lasting about ten minutes. On the night of the third day her attack only lasted five minutes, while the following night she had no attack at all, for the first time in 20 months, and during the day she had been able to walk up six flights of stairs without stopping, on four different occasions,—a feat that she had not been able to accomplish for a very long time.

After seeing this case I am the first person to admit that chronic painful symptoms connected with a vascular disorder and having more or less of a resemblance to angina pectoris may yield promptly to the influence of a few doses of Trunecek's serum; on the other hand, in my case no modification occurred either in the condition of the aortic valves or in the aneurism of the carotid artery. For these reasons I wonder whether it is really possible that any decrease in size of the large vessels can be produced, since their mensuration is very difficult, and there is no strictly accurate method of verifying the fact. A radiograph would be about the only thing that could settle the point. I therefore consider the question as still open until new cases, examined by unquestionable methods, shall be published.

The inorganic serum also relieves the dyspnea of arteriosclerosis. Trunecek calls attention to a case of arterial sclerosis with cardiac hypertrophy in an old man of 74 years, in which only three injections were used, the dyspnea improving from the beginning. A woman of 33 years, with arteriosclerosis and anemia, found that her dyspnea disappeared completely when 5 c.c. of the serum had been injected. The most striking point in the facts that have just been cited is the small quantity of serum that was administered.

François-Franck tells me that he also has seen dyspnea in patients of this order yield to a few injections of serum. The following case he cites as an instance: Mme. X., 56 years of age, with arteriosclerosis, and an emotional condition, has suffered from vertigo and oppression with attacks of dyspnea which are particularly noticeable during the periods when she does not urinate much. The iodid treatment gave no relief, and she was then put for two weeks on injections of Trunecek's serum, beginning with 1 c.c. and increasing to 2.5 c.c.,—eight injections in all. Although she herself did not report any change in her usual method of breathing, the following fact revealed the improvement that had been effected: She lived on the fifth floor of her house, but had only gone up so high because there was a lift. This lift got out of order a few weeks before the beginning of the serum treatment, and, feeling that she would be unable to walk up to her apartment, she had taken refuge with a friend. During the last three days of the new treatment the elevator again got out of order, and this time the patient paid no attention to the fact, but walked up the five flights without being more out of breath than another person would have been after such a climb.

I for my part can mention four cases in which oppression yielded to this treatment. The first is that of a young woman, nervous, subject to sudden congestive attacks of the face and ears, and whom I am specially treating for ringing of the ears and deafness. One of the first symptoms whose disappearance struck her was the shortness of breath she noticed when walking, and which she no longer felt after the beginning of the treatment. At the present time she can take long walks without feeling this symptom.

A nurse of Mathieu's ward, whom I am taking care of for auricular symptoms, noted that together with the disappearance of her ear noises the dyspnea which she usually felt on going upstairs ceased.

A saturnine patient, with arteriosclerosis and arterial hypertension, with albumin in the urine, found that after a few injections both the palpitations that he had had every day even when seated or lying down for the previous 18 months disappeared, as well as the attacks of oppression from which he had suffered during the same period.

A woman of 54 years, with long-standing syphilis, complained of oppression at night occurring whether she was lying on her right side or on her back. To this she had been subject for six or seven years. Any sudden movement would bring on the dyspnea and she also had great difficulty in going upstairs. Two enemas of serum, or 25 c.c., lessened these symptoms in a remarkable manner.

Marmisse reports in his case complete relief from disagreeable attacks of cardiac asthma.

The action of the serum is also evident in arterial cachexia.

Trunecek relates the case of a man of 59 years, who had formerly had the alcohol habit, as well as an attack of apoplexy, and who was seized with suppurative arthritis of the foot with ankylosis of the knee on the left side; there was also a very marked condition of cachexia. In two months' time, when 16 c.c. of serum had been administered, sleep and appetite had returned, the general condition had improved, the foot and knee had noticeably decreased in size, while later on all the fistulæ of the foot closed. When 58 c.c. had been administered the patient began to walk, and the knee and foot resumed their normal dimensions.

Marmisse's case, which is very similar to the foregoing, may be mentioned here. It was that of a man of 86 years, suffering from arteriosclerous cachexia with manifold manifestations. In addition to cessation of the attacks of asthma, and to decrease in the dyspnea, the writer notes the disappearance of cyanosis, return of physiologic heat, sleep and appetite, increase in muscular force, and improvement in spirits. On the other hand, no benefit was seen in the ulcerations of the foot, as had been the case with Trunecek's patient.

Such were the facts published by Trunecek, and corroborated by comparative cases. They deserved to be verified, and formed the point of departure of my researches. I first took up the subject of cerebral arteriosclerosis, and I chose patients who showed the disorder in the form of lasting impairment of function, localized particularly to one side of the body. The symptoms present were both of a sensory nature (paresthesia, tingling, and numbness) and motor (paresis rather than paralysis, disordered walk, etc.). Reflexes were increased, and Kernig's sign was habitual. Epileptoid trepidation of the foot was found in some of them.

In two cases of this form of cerebral arteriosclerosis, sensory-motor hemiplegia with lasting impairment, I found that in one, in whom the symptoms had begun two years previously, after 20 injections, making a total of 57 c.c. of mineral serum, the tingling which he felt in the face and foot disappeared, that of the hand decreased, while the muscular force of both arms and legs was improved. The patient, who could not walk 50 yards without sitting down when I began, could now walk all day.

In the second case, which was treated at the same time, the numbness of the foot decreased noticeably; the patient became able to walk indefinitely, his hand became more skilful, and his attention improved. He can now carry out his work as a draughtsman, after having given it up for several months.

I then found that if the pathologic phenomena, instead of being already chronic, were of sufficiently recent origin, the results obtained were better still, since in a third case, in which 50 c.c. were injected, I found that the tingling disappeared, strength returned to the hand that had lost it, ear sounds ceased, while memory and sight improved.

Another patient enabled me to ascertain the efficacy of the serum in cases in which an acute complaint had brought on, in an arteriosclerous subject, symptoms of cerebral monoplegia.

Trunecek's serum does not act only on localized symptoms, but has a favorable effect on those of a more diffuse nature, such as vertigo, disordered sight, ear sounds, headache and memory defects.

I cannot at the present time dwell on instances that I have furnished as evidence of these facts, and which will be found in my paper on the subject; I only wish to mention the complete disappearance in one of my patients of a constant state of vertigo which had lasted six months.

I also studied the action of the serum in cases of complete hemiplegia following a stroke, and dependent on arteriosclerosis.

Without dwelling on cases of recent hemiplegia, which are not demonstrative, I published the case of a woman of 51 years, suffering from incomplete left hemiplegia with paralysis of the movements of elevation of the eyes, and of power of convergence, and who after ten months of treatment by the serum now walks without a cane, whereas at first it was difficult for her to leave her bed and impossible for her to walk without help.

Since the publication of the paper in which all these facts are related in detail I have treated further cases of the same type. I shall mention two of them, particularly as they showed Babinski's sign, which had been lacking in all the others.

A man of 66 years, suffering from pseudobulbar paralysis, with vertigo and tendency to fall to the right, had his vertigo and hemiplegic symptoms improved by 20 injections of mineral serum varying from 2 c.c. to 5 c.c. each.

A woman, whose main trouble was a crural monoplegia with great difficulty in walking, rapidly recovered the strength of her paralyzed limb. I regret to say that I have lost sight of this patient, particularly so as she was suffering from glaucoma and had noticed after a few injections favorable though slight changes in her vision.

As regards cerebral arteriosclerosis, I can also mention the cases of Thiroloix and of Goldschmidt, of Strassburg. The former treated a lady of 60 years, whose father died at 77 of cerebral hemiplegia, and who herself had been subject to migraine and epistaxis between the ages of 15 and 25 years. Since reaching the age of 45 she had suffered from pains in her joints. Since the age of 53 she had shown signs of cerebral and renal arteriosclerosis, complaining of vertigo, defective memory for recent facts, and dyspnea; there was furthermore an irregular pulse, slight edema, and a systolic murmur at the aorta. Injections of serum were made for two months at doses of 2 c.c. a day. All the symptoms improved, but the fact that struck the patient's relatives more than anything else was the return of the psychic faculties and memory. The vertigo disappeared almost entirely, the edema of the legs was absorbed and did not reappear, and improvement was permanent. It should be noted that neither milk diet nor iodid had been able to produce this result.

Goldschmidt's case concerns a man of 66 years, who in 1895 had a slight attack of left hemiplegia and in whom signs of progressive paralysis appeared after great intellectual fatigue. The patient slobbered whenever he tried to speak, and had difficulty in swallowing. He had frequent desires to urinate and was not always master of the act, his walk was slow and stiff, the joints were less movable, there was progressive deafness, reflexes were present, and skin sensation was intact. After 20 subcutaneous injections, first of 2 c.c. each, and then of 3 c.c., 4 c.c., 5 c.c., and 6 c.c., he was found to be much wider awake and gayer, while he swallowed and walked better. There was greater freedom in the joints, and greater control over the bladder, but salivation was still abundant and his speech was jerky and stiff. In a word, the case showed improvement that was appreciated both by the patient and by those about him.

It is therefore manifest that cerebral arteriosclerosis can be improved by the concentrated serum treatment, both in its localized and in its diffuse manifestations. It is well, on the other hand, to have clearly understood that when there are signs of destruction of tissue, the treatment will give no result. In order to make sure of this I treated by injections a young patient who had had total aphasia for four years; the injections, kept up for two months, were followed by no alteration in the patient's condition.

The treatment is equally powerless in cases in which hemiplegia is caused by a spot of necrosis due to a cardiac embolus, as is shown by the following case: A young woman of 30 years, with mitral disorder, and with right hemiplegia without much contracture or Babinski's sign, derived no benefit whatever, except that her strength improved, from nine injections of 1 to 3 c.c. each made for nine consecutive days.

It is therefore important in order to obtain a favorable result to be careful in choosing the subjects to be treated, and not to undertake a case until it has been thoroughly examined. As regards paralysis it is in the forms in which hemiplegia occurs without stroke, and in which the sensory signs are equal to or exceed the motor, that it is possible to expect with almost certainty, if not recovery at least marked improvement, and the more recent the case the more marked the improvement to be expected.

I think that there is a new and efficacious application for the serum in sclerotic otitis. The disappearance of ear sounds in one of my patients, the removal of a constant state of vertigo in another, and the improvement in hearing of a third, suffering from aortic regurgitation, albuminuria, and retraction of the palmar aponeurosis, have led me to treat systematically by this method the symptoms of sclerotic otitis. Twenty-two such cases have passed through my hands and will form the subject of a special publication when time shall have shown whether the improvement lasts.

In 14 cases there were signs of more or less marked deafness; one of my patients did not hear his cuckoo clock, although he got on a chair to do so, and another had to have words shouted into his ears. In five cases there was ringing without deafness, and in seven both were present. It can be said that improvement was obtained in almost all cases.

As regards the ringing, it usually disappeared gradually. The improvement in deafness was sometimes so rapid that after the first injection the patient could hear sounds that had been lost to him for years; this varies, however, with the patient's age, degree of deafness and arterial elasticity.

When there are both ringing and deafness, the ringing yields first, before the hearing improves noticeably; at least this is the impression that the patient has of the occurrence. In the cases of Thiroloix and Goldschmidt, improvement in hearing was also noted.

There is therefore here an entire series of cases which, in spite of their clinical divergence, really belong to a single group, and have as their anatomic substratum hardening of the arteries.

In another paper I shall discuss the way in which the serum acts in arteriosclerosis, which will lead me to consider its use in other morbid conditions, such as rheumatism and nervous complaints; I shall, in accordance with the mode of action involved, study the question of what can be expected from this treatment, particularly in congestive conditions; and finally, I shall compare Trunecek's serum with other concentrated serums, both artificial and natural.

PRACTICAL NOTES ON THE PROPHYLACTIC AND CURATIVE TREATMENT OF INFLUENZA, MALARIA, ERYSIPELAS, AND OZENA

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INFLUENZA

During recent years it has become a common practice to designate an old-fashioned "bad cold" as the influenza or la grippe. This is a disorder that has devastated the world since the earliest times. Livy mentions an epidemic allied to it in character occurring as early as 412 B.C.; Noah Webster and other writers describe diseases having the same character occurring in the early part of the nineteenth century; and some of the older physicians, who are familiar with the history of the early forties, will recall the disease as the Tyler grippe, from its prevalence during President Tyler's administration. At that time it assumed the character of an epidemic and then, as now, it was attended by fatal results in many cases, particularly when pneumonia and other serious complications supervened.

Influenza is now known to be due to a micro-organism—the bacillus of Pfeiffer. It is therefore infectious; but it is not contagious in the ordinary sense of the word, though it may be communicated from the sick to the well in a closed apartment. For this reason isolation is a preventive. Patients, however, should not be kept in one place—they should be given plenty of fresh, particularly country, air. As a preventive and to enable those exposed to the infection the better to withstand the disease, the bacillus should be searched for in sanitariums, and Boards of Health should insist upon proper ventilation of large stores, auditoriums, and dwellings. It is doubtful whether there are better predisposing factors than over-work and over-heat. Conversely, relaxation and ventilation are preventives.

During the prevalence of an epidemic scarcely a family in the infected district escapes the disease. But individuals who practise proper prophylaxis may escape, and those attacked may rob the disease of many of its dire consequences by prompt resort to treatment. At the onset of the disease, at the first manifestations of chilliness or general malaise, a hot foot bath followed by a glass of hot lemonade may be advantageously taken. The hot lemonade may be continued with good service. The following formula has been found efficacious in many cases:

℞	Mercurous chlorid (calomel)	6 grains	0	4
	Quinin sulphate	30 grains	2	0
	Powdered ipecac and opium	20 grains	1	3
	Powdered camphor	12 grains	0	8

Mix and divide into twelve capsules.
S.—One capsule every two hours.

In some cases these capsules may have to be discontinued on account of nausea induced by the Dover's powder. In such cases the following may be substituted with good results:

℞	Caffein citrate	5 grains	0	
	Quinin sulphate	18 grains	1	2
	Phenacetin	60 grains	4	0
	Powdered camphor	20 grains	1	3

Mix and divide into twenty capsules.
S.—One capsule every three hours.

Should the symptoms abate, the capsules may be administered at longer intervals, or they may be discontinued for a time.

The foregoing treatment will be found not only to mitigate the severity of the symptoms of the disease, but it will also lessen the likelihood of the development of complications and sequels. The importance of prompt treatment with a view to prevent the occurrence of complications is evident from the fact that according to official statistics 51,278 persons died of influenza in Prussia from 1890 to 1894.[1] Over one-half of these deaths occurred in persons over 60 years of age, and most of them were due to pulmonary complications, such as bronchitis and pneumonia, which in many cases are preventable by resort to prompt and thorough treatment.

MALARIA

Malaria is a disease that is now known to be due to infection of the body with a special micro-organism known as the plasmodium or hematozoon of malaria. Formerly there was much wise talk of miasms and of the disease being due to the emanations of a pestiferous nature from the lowlands of a marshy region. We now know that the disease is transmitted from one person to another through the agency of mosquitoes—a mosquito becoming infected through biting a malarious subject and subsequently infecting another person when seeking to quench its thirst for blood. The undoubted relationship of lowlands, marshy regions, and stagnant pools to the disease is now explained since it has been ascertained that these serve as breeding ground for the particular species of mosquito—the Anopheles—that transmits the hematazoon of malaria.

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[1] Quoted by Tinkell, Twentieth Century Practice of Medicine, vol. xv, page 242.

The remedies that we depend upon for the cure of malaria are quinin, arsenic, and mercury. The depurative effect of mercury, especially its effect on the liver, and the alterative effect of arsenic are important adjuvants to quinin in many of the chronic cases; the antiperiodic effect of quinin, however, is manifest independently of preliminary medication with these remedies. In this connection I wish to emphasize the great value of a single large dose of quinin, thirty grains, combined with a half grain of morphin, given one-half hour before the expected malarial paroxysm. This is distinctly prophylactic as well as curative treatment; it serves to break up, to prevent, the paroxysm in all varieties of intermittent malarial fever, and it is curative in that no recurrences occur. For a long time I have insisted on the great value of this form of treatment which has most satisfactorily effected a final cure in all cases of intermittent fever in which I have used it. I have been especially impressed with its value in cases of long standing. In my own person the treatment was adopted without previous medication to enable me to meet an appointment that would have been frustrated by the chill and fever of the tertian intermittent fever which was approaching. A complete jugulation of the paroxysm followed and there has been no recurrence after the lapse of many years. An important result of the treatment is that, having cured the malarial subject, it enables him to remain in the district where he contracted the disease, without the likelihood of recurrences. This I have repeatedly observed. I have no doubt that the severe disease prevalent in many sections of the South and known as congestive fever may be most effectually combated with this combination of quinin and morphin. The treatment should be instituted immediately the diagnosis is made, even at the onset of a paroxysm, without waiting for the cold stage.

ERYSIPELAS

Erysipelas is readily diagnosticated—requiring only the sight and touch of the skin surface involved to decide the nature of the disease. It is a disease also that may be readily controlled by efficacious treatment which if instituted will prevent the widespread destruction of tissue so common after ordinary methods of treatment. The treatment that I advocate consists of the hypodermic injection into the erysipelatous area of a mixture of carbolic acid one part, glycerin three parts, and distilled water four parts. One half dram of this should be injected daily into each six inches square of the erysipelatous area. This should be done at three sittings at intervals of four hours, not more than two inches square being injected at a sitting. Sometimes the skin of the erysipelatous area is so hard that it is necessary to introduce the needle at the margin of the indurated area. In these cases, however, the arrest of the disease is as complete as when the needle is introduced directly into the inflamed area. This constitutes all the treatment necessary to arrest the disease in any part of the body. I have used it in many cases and have found it far superior to measures, such as encircling the skin surface involved with iodin or nitrate of silver which, in my hands, have proved of no avail. I believe also that it is of value in preventing recurrences, as I have used it in many patients previously subject to recurring attacks of erysipelas and in none has recurrence subsequently followed.

OZENA

Ozena or atrophic rhinitis, the late stage of a chronic nasal catarrh, is an intractable disorder, and that its most distressing features be obviated, it should receive attention in the formative stage. In an advanced stage an offensive odor is a notable feature, but in addition a distressing symptom and the cause of much irritation are the incrustations that form on the mucous membrane. The condition may be much relieved by the use of the following:

℞	Spirit of turpentine,		
	Olive oil, of each	2 ounces	60|0
	Camphor	2 drams	7|5

Mix, and apply throughout the nasal cavities, using a sponge, probang, or the little finger.

This mixture is indicated in all stages of the disorder and should be applied sufficiently often to keep the mucous membrane clean and free from incrustation—three or four times within the twenty-four hours.

In the early stages douches of sodium chlorid, one teaspoonful to a half pint of water may be used with good effect. Though this is in general use, I prefer a douche of boric acid. In most cases, however, I use boric acid in a manner that I believe is original. Knowing that much of a powder introduced into the nose by insufflation finds its way into the throat and upper air passages, and knowing that in the act of blowing through the mouth the subject closes the soft palate, it occurred to me that a general diffusion of a powder through the nasal cavities might be effected by requiring the patient to blow it from the front. I therefore introduce one end of a flexible tube into the patient's mouth, and the other end filled with boric acid into his nostril; then directing him to blow, the powder is diffused throughout the nasal cavity and most of it does not as it otherwise would find its way into the throat. I have found this treatment much more efficacious than the use of a boric acid douche or spray. This insufflation should be carried out every morning, and may be followed by the use of the following spray:

℞	Carbolic acid	1 dram	4.0
	Glycerin	3 drams	12.0
	Distilled water	4 drams	15.0

Mix. S.—Dilute in the proportion of one dram to an ounce of water and use as a spray.

If this treatment is not followed by a complete cure within a reasonable time I am in the habit of using a mixture consisting of equal parts of alum and tannic acid thrown into the nostril by the aforementioned flexible tube. As a last resort, I use a mixture consisting of powdered nitrate of silver, five grains, and powdered gum arabic, one dram, thrown into the nostril in the same manner. Of course, the milder remedies should be persisted with before recourse is had to the more severe.

In addition to the local treatment, general treatment is indicated in all cases. The gastrointestinal tract must be kept in an active condition by regulating the diet and by the use of mild cathartics, if necessary. Different alterative and tonic preparations should be used, and these may be combined with or be followed by the syrup of the iodid of iron, one half teaspoonful three times daily, for two or three weeks. As a general tonic some of the organic preparations of iron, and preparations of iron and manganese, will be found of much service.

While mild cases may be expected to yield to the treatment within a short time, severe and advanced cases will probably require the continuation of the treatment for a long time. Persistence, however, will usually be rewarded by complete exemption from distressing complaints.

A RECENT ADVANCE IN THERAPEUTICS: LOCAL TREATMENT

BY CHARLES BOUCHARD, M.D.
Professor at the Paris Faculty of Medicine

The idea occurred to me that, since a general treatment has a specific curative effect as well in local diseases as in general diseases that become localized, an effort might be made to limit the action of the remedy exclusively to the tissue that is affected by injecting into the disordered region the remedy that is efficacious when it is administered to the entire system.

In cases of acute articular rheumatism, a man weighing 60 kilograms (132 pounds), to whom 6 grams (90 grains) of sodium salicylate are given each day, finds that, either simultaneously or in succession, the swelling of each joint disappears. Ten centigrams of the remedy have been administered each day to each kilogram of his weight, to each kilogram of healthy tissue, as well as to each that is diseased; if, now, in each joint (to speak only of the larger ones) the soft tissues that are diseased weigh between 50 and 100 grams, the recovery of each local lesion is brought about by doses of from 5 to 10 milligrams.

If clinical experience were to justify this conception, to give to a man in a day 6 grams of sodium salicylate for a rheumatic inflammation of a single joint, would amount to supplying the diseased joint each day with 10 centigrams of medicine that is necessary and required, and to furnish the remainder of the body, which has no need of them, with 590 centigrams of a substance which is certainly useless, even if it is harmless.

Facts show that an acute rheumatic arthritis yields to a local injection of extremely small quantities of sodium salicylate. I do not claim to have cured them with 1 centigram (one-sixth grain), but I have seen cases of arthritis stopped off short with 3 centigrams (one-half grain); and it is unusual if an articular inflammation cannot be cured with 10 to 20 centigrams (1½ to 3 grams) dissolved in 2 to 4 c.c. (30 to 60 minims) of water.

It must not be thought that this is merely a case of counter-irritation, as when distilled water is injected hypodermically; in the foregoing degree of concentration the solution used is not painful. It should also not be thought that this effect is obtained by the removal of liquid from the tissues, as happens when concentrated saline solutions are injected; recovery is obtained just as well when care is taken that the solution is absolutely isotonic, when it has the same osmotic tension as the blood, when it congeals at 0.56° C., as is the case with 3 per cent. solutions of sodium salicylate.

A patient of mine, suffering from rheumatism of the knee with a chronic tendency, had been in bed for two months, and had in six weeks derived no benefit from my attempts at treatment, both general or local; but 10 centigrams of salicylate given in a single injection enabled him to get up the same day, and on the following day he was cured.

There next came under my observation a series of cases of acute polyarthritis, both with fever and without, in which I found that 5, 10, or 20 centigrams of salicylate put a stop to the redness, pain, effusion, and immobility; and this favorable effect occurred only in the joint that was treated. The other joints were not modified, and seemed to be ignorant of what was taking place near them, as well as of these results. Other joints, still, may become inflamed, but they do not influence the one that is getting well, nor are they influenced by it. You can thus cure any joint you like, while the others remain, or may become, inflamed. This proves that the minute doses injected locally have no general action, and that the local recovery is not due to some general influence on the blood or on the nervous system; it also proves that when a general treatment is efficacious, each joint taken by itself recovers, not through the total amount of the drug that is spread throughout the system, but through the very small amount that is furnished to that particular joint.

Though recoveries by means of local injections of very small doses are often final, they are sometimes not so. But in such cases there is nothing to prevent the local treatment from being continued or begun again, just as nothing prevents several joints from being treated simultaneously or in succession.

Although the local treatment of a case of acute, or erratic, articular rheumatism is efficacious, it must not be considered sufficient.

It no doubt reduces the temperature when only one joint is affected, but it does not prevent the inflammation of other joints, nor the large serous cavities from being affected. In such cases the general treatment is obligatory; if it turns out to be inadequate, the local treatment will come to its help.

When an attack of rheumatism is on the decline, when it no longer exists as a general disorder, and has only left some lasting traces, and especially when it is a local affair from the outset, the local treatment may be found sufficient.

It is not inefficacious even in chronic forms, when its effect is useful and manifests itself sometimes with a rapidity that exceeds belief; but it must be applied with perseverance to have a reasonable expectation of obtaining a lasting, functional improvement.

It is, of course, understood that I am only speaking of true rheumatism, and not of the class of infectious forms of false rheumatism, gonorrheal in particular, although the latter is relieved by the local salicylate treatment, so far as the pain is concerned. In several cases an injection at the painful spot in the wall of the chest has stopped and cured pleuritis at the outset. Even in rheumatic pericarditis, I have seen an injection of 10 centigrams bring the temperature down from 102° F. to normal; a second injection was then made, and in three days all rubbing sounds had disappeared. In endocarditis I have failed.

By this local salicylate treatment I have improved and cured painful muscular contraction of the abductor muscles of the thigh by acting on the insertions of the tendons. I have also obtained some good results in sciatica, as also in the severe and obstinate neuritis of herpes zoster. I do not inject the remedy into the tendons, nor into the nerves, but in the neighborhood of the disordered tissues; in the same way, in inflammation of the articular or visceral serous membranes, I do not introduce the remedy into the closed cavities, but into the surrounding tissues, as near as possible to the disordered parts, or even directly into them, when feasible.

The successful results that I have just mentioned led me to ask whether it might not be advantageous to apply the local salicylate method to other painful complaints of the nerves, and I began the experiment with the lightning pains of tabes. But I obtained no results at all, nor has Professor Déjerine, who, at my request, also made the experiment. Just as the salicylate, which cures true articular rheumatism, has only a dubious action on gonorrheal arthritis, so also its efficacy in certain cases of neuritis, that may be of a rheumatic nature, does not appear when the remedy is applied to nervous pain like that of tabes, which is due to a specific cause other than rheumatism. There was, therefore, a manifest indication to try another specific remedy, and I experimented with 3 per cent. injections of potassium iodid; solutions more concentrated than this give rise to considerable pain. These injections, made into the painful nerve itself, or at the points where the nerves first make their appearance, or even at the spots whence the pain seemed to start, resulted in improvement, even of a permanent nature, but they did not seem to accomplish more than a given amount; the pain became bearable, but it was still there. I did not use more than 2 c.c. per injection, or 6 centigrams of iodid.

I then decided to combine biniodid of mercury with the potassium iodid. My solution contained 1 centigram (one-sixth grain) of biniodid of mercury and 4 grams (60 grains) of potassium iodid to 100 grams (3 ounces) of water; never more than 2 c.c. were injected at a time; in other words, two-tenths of a milligram of mercurial salt; consequently, my doses were extremely small. But in this way I overcame some most severe and stubborn forms of pain, although I cannot yet say how long these happy results will last.

My success in these cases led me to try a local treatment for the local manifestations of syphilis by means of the same tiny doses of specific remedies. I have not yet had time or occasion to assemble so many cases as in dealing with rheumatism, but I will mention those that I have seen.

A man suffering from an ulcerated gumma of the side had been treated by the two specifics, first separately and then by the two combined; he had been put on inunctions and on hypodermic injections of mercurial salts made in the customary spots some distance away from the lesion itself. But as the general treatment did not succeed, I stopped it, and decided to make in the neighborhood of the gumma injections first of 1 c.c. and then of 2 c.c. of potassium iodid. After only two injections, one of 3 and the other of 6 centigrams of potassium iodid, the gumma was healed. Under the same conditions, that is to say, after failure of general treatment and its discontinuance, the gumma in another case disappeared under the influence of three injections of potassium iodid. In another patient, suffering from neuritis due apparently to the same specific cause and accompanied by trophic ulcerations, the signs of neuritis disappeared, and healing of the ulceration was effected by the same injections of tiny doses of potassium iodid, although they then had to be kept up for a longer period.

A woman afflicted with a serious form of rapid syphilis showed on different parts of her body deeply ulcerated condylomas. I put her on a general treatment by the two remedies, which were administered in the different ways by which they can be introduced into the system. At the end of a month's time absolute intolerance of the remedy appeared; the stomach could no longer stand the iodid, while injections of benzoate, and afterwards of cyanid, of mercury gave rise to indurations the size of a walnut. As no improvement had been produced, and as the general treatment had shown itself to be useless and harmful, I stopped it, and tried on some of the growths the local treatment by means of tiny injections of potassium iodid and biniodid of mercury in the doses mentioned above. In three days one of the tumors, into the base of which two injections of two-tenths of a milligram of biniodid had been made, had healed. Another ulceration yielded after six similar doses, but during this time the other tumors, which were no longer held in check by any general treatment, did not improve, while the patient's weakness increased and her hair began to fall out again.

In this instance the result of the local treatment shows clearly what the latter can accomplish, and what we have a right to expect from it. Even in the absence of any general treatment, it cures what that treatment failed to cure; but it is without influence on the general condition, and does not check the progression of other local lesions that are not treated, nor the reaction on the entire system of the disorder when left to itself. As was the case with rheumatism in course of evolution, syphilis in course of evolution requires a general treatment. I even think that it requires it in all cases. But with or without general treatment, we shall be able, I hope, by means of local treatment to gain the mastery over a lesion that is isolated or has come to a standstill, and quickly to put a stop to certain very serious, painful, or dangerous localizations of the disease that occur on the face and in the tongue or eye. There seems, indeed, no reason why we should not hope some day to be able to master lesions that are more deeply seated.

I do not think it will be necessary for me to point out the way to follow if any one is desirous of applying this method to the treatment of other diseases.

The efficacy of local therapeutics by means of very small doses appears to me demonstrated. As to the way in which it acts, the first idea that occurs is that it is an offshoot of antiseptic therapeutics, and that it acts locally on the causes that give rise to the disease rather than on the animal cells in their struggle with these causes. First impressions are not always bad; but, although this interpretation of the facts is natural, I must admit that it is not at all proved.

The objection can be made that the amount of the drug is too small to have any antiseptic action. Ten to 20 centigrams of sodium salicylate is certainly a small amount of an antiseptic substance, even if we grant that it is confined in the 10 to 50 grams of living tissue affected by the morbid process in a case of rheumatic arthritis. Still, the proportion amounts to from 2 to 20 per 1000, and no one can say that this is not a sufficient strength to influence the cause of rheumatism, which may be a microbe more sensitive than others to the action of this salt, which may happen to owe to this circumstance its specific effect in rheumatism. As to the doses of two-tenths of a milligram of biniodid of mercury in a small condyloma, this might represent a proportion of 5 to 10 centigrams of mercurial salt per kilogram of diseased tissue, which is a proportion that is supposed to be certainly antiseptic for the majority of microbes, and that is infinitely greater than the amount of silver required to check the growth of the aspergillus.

The old objection may perhaps also be renewed, which claims that antisepsis is impossible because the dose of poison necessary to kill a microbe will be much more likely to kill a nervous cell. Although this objection has a certain semblance of foundation when applied to general antisepsis, which spreads the remedy equally throughout all the tissues, it cannot be invoked as regards local therapeutics. If 5 grams of sulphate of quinin were to be spread throughout all the tissues of a man weighing 50 kilograms, he would have in each kilogram of his body, and consequently in a kilogram of his nervous substance, 10 centigrams of the drug, which might be enough to occasion death; but it is possible to place the same drug in the cellular tissue in the proportion of 1 to 5, which is 2000 times stronger, without killing, or even compromising, the vitality of the elements of that tissue.

Local therapeutics, therefore, owing to the fact that the action is local, enables us to place the drug in the diseased tissue in proportions that are really antiseptic, without any danger that its ulterior diffusion throughout the entire system may render it toxic.

The happy results obtained in my attempts to effect a local form of therapeutics by very small doses are possibly due to some antiseptic action; it is not impossible, however, that there may be, as well, some calling forth of the natural means whereby the system of an animal defends itself from infection.

The method of local injections of small doses of drugs is not dissimilar to the many instances in which the surgeon comes to the help of the physician and effects a local cure of various disorders. Thus, we have only to take as an example the local treatment of lupus. What a lamentable account could be written of the drugs that have been used to no purpose in the general treatment of this local disorder, which yields to local treatment. More lamentable still would be the calculation of the cost of iodin, cod-liver oil, and other expensive and harmful substances, that have been used in the treatment of tubercular abscesses and gummas, until the day when, having ascertained the local cause of the disorder, physicians made up their mind to call the surgeons to their aid; this is equally true of tuberculosis of the lymph-nodes, and even of tubercular peritonitis.

The oculists have preceded us in this line for many a day, and have used specific or non-specific remedies in the nearest proximity to the diseased tissue; their example might have been followed much earlier with advantage.

The same idea holds true for the nasal fossæ, where local treatment of coryza often prevents the spread of the infection to the entire respiratory tract.

The realization of intestinal decomposition and of gastric fermentation led to the method of local antisepsis of the digestive tube, the good results to be derived from which I made known a number of years ago.

It is hardly necessary for me to recall the wonderful results that followed the bold idea of carrying even into the depths of the brain a local struggle against a local disorder, the exact seat of which we had learned to ascertain.

Time and space would be lacking if I were to endeavor simply to name over all the conquests of local therapeutics. The domain of this method is not limited to infectious disorders. I have already mentioned the oculists, who not only bring mercury and iodin into contact with the diseased ocular membranes, but instil near the morbid focus atropin, eserin, and pilocarpin. In the same way, in internal medicine local therapeutics is not specific only; it can be physiologic as well.

Thus, ergot given internally, or a pyocyanic culture injected beneath the skin, checks hemorrhage; their general action is opposed to loss of blood resulting from local vascular lesions, and all the vessels of the system are tightened up in order to produce that effect on a single vessel. But there are substances which when applied locally produce ischemia, and, when desired, hemostasis. Thus, cocain applied to the surface of the nasal membrane checks epistaxis almost instantly, even those serious forms of this disorder which in cirrhosis are caused by the erosion of an artery in some part of the septum. I have observed a few drops of a solution of adrenalin cause, by simple superficial application, shrivelling up and indolence of painful hemorrhoids. On two occasions I have seen 1 c.c. of a 1 to 10,000 solution of this substance stop serious hemoptysis after being injected into the trachea through the neck.

It is childish and uncertain to wish to play the part of a prophet, and I shall not endeavor to foretell what the therapeutics of the twentieth century is likely to be. I feel convinced that our successors will discover ways that we do not even suspect to-day. But what I can see is the direction into which the art of therapeutics appears to be entering at present. It does not set aside all general action on the whole system, but accomplishes it by the use of chemical substances, and especially by the application of physical agents. It also acts on the nutritive process, either when nutrition alone is concerned, or when disordered nutrition appears to favor the development or continuation of infection. It resists this infection by general means, but particularly, and more and more, by local means, whether it exerts some antiseptic effect, or whether it arouses an antitoxic reaction; and through these means it can be considered a true healer of disease. But, above all, it is guided by the notion of infection, which was the glory of the nineteenth century, and which the twentieth will continue to develop and perfect.

THE REST TREATMENT: WHEN INDICATED AND HOW CONDUCTED

BY JOHN MADISON TAYLOR, A.M., M.D.
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Treatment by rest, isolation, and forced feeding, as taught by Weir Mitchell, has had many advocates who meet a wide variance of success. It is interesting to scrutinize the reasons for both the good and the poor results obtained. The system has so much to recommend it, it is so eminently a rational measure viewed as a whole, it is adapted theoretically to so large a variety of conditions, and it is capable of such endless modifications without impairing the integrity of its unity, that it is strange more medical practitioners do not oftener adopt its essential features. The so-called failures are in most instances capable of intelligent explanation, and would have proved successes had certain precautions been taken —precautions which would have been accepted by the physician had their significance been noted in time.

It has been my privilege to see the proof of this during the years of my association with Dr. Mitchell, and to note abundant evidences of the truth that a master workman can get satisfactory results with the same material even after the discomfiture of imitators who yet enjoyed the fullest opportunities. It is not always easy to accomplish this—for reasons pointed out by me elsewhere.[1]

The value of the measure is to be estimated not so much upon the reasonableness of mere rest, isolation, and forced feeding, but upon the collateral factors differing with each case. These are ever the same in principle, but they vary with the circumstances under which the process is begun, and the amount of authority permitted the physician for the exercise of control over the freedom of action of the patient. Above all final success cannot be expected unless caution is exercised in slowly but judiciously resuming customary activities and responsibilities.

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[1] Repair of Will Loss, By the author. International Clinics, Vols. II, III, IV, 7th Series.

In all this the wisdom of the medical adviser is tested, or perhaps to be more exact, his genuine interest in the welfare of the patient is put to the touch. It may be no fault, only a permissible peculiarity, in the physician to feel a deeper interest in the clinical features or pathologic state of the patient rather than in his rate of progress towards health. Not only so, but a state of long continued ill health, or a series of ailing conditions, renders an individual unattractive or positively irksome to the family adviser; whereupon it is well to place him for the time in the hands of some one else who can, and will, use every effort to secure as far as possible relief or cure. This other physician need only to be wise and of practical good sense, not a neurologist. Again, it sometimes happens that a neurologist exhibits his highest and most estimable qualities when he furnishes a correct diagnosis and outlines the treatment. As a rule he is too busy to descend to details of sleep, baths, food, outings, amounts and forms of exercise, gradations of employment, itineraries, and the like. This the general practitioner can and should do. Even the sculptor thinks his work almost done when he finishes his sketch and turns over to the skilled workman the actual cutting of the marble block.

The dominant note of the rest treatment lies in the fact that the nerves of the neurasthenic being slowly and thoroughly tired, all the tissues of the body, including also the brain cells, need to be put as nearly out of commission as possible, to secure slow but certain structural and functional repair. To effect this it is not enough to place the patient in bed and outline a series of measures calculated to return health to one suffering from a condition of acute illness; but, in dealing with the mentally jaded, it is imperative to erect a barrier to those powerful agencies for harm of a psychic sort, among which are to be numbered the knowledge and discussion of the ordinary affairs of life. The physician often fails to appreciate the fact that in the circumstances of the life of every adult, young or old, there is much of a nature calculated to aggravate the condition of over-wrought reflex irritability, called nervousness. Hence isolation is of paramount importance in most cases of persistent ill health, in which the psychic and emotional element is the dominant factor. The isolation must be, in extreme cases, complete. After this has been long enough maintained it may be gradually and carefully omitted. Again, hyperalimentation is needed, because long-continued ill health inevitably brings about destructive depletion in those cardinal paramount structures, the nerves. And these, as is known by those who search for fundamental truths, are ever slow to repair. If the fact be lost sight of that in states of exhaustion, bodily tissues regain tone in very different degrees of rapidity, fatal mistakes will be made and failures, due to the fault of the medical adviser, will certainly result. The principle is well established that much time and wise careful readjustments are required to overcome the dangerous exhaustion of modern life, hurried and harassed as people are by the competitive struggles of existence. Mitchell approached the problem with a thorough knowledge of physiology of which he was a qualified exponent. It is only of late years, a decade or less, that the physiology of the nervous system is being recognized as the paramount factor of clinical knowledge, and through this alone a clear appreciation can be had of the complex disorders which baffle medical skill, and result in chronic invalids, dilapidated and blighted in both body and mind.

It is now demonstrated that there resides in nervous tissue a unique potentiality closely analogous to electrical energy which constitutes a storage battery of nervous forces, motor and other, varying under different circumstances of nutritive vigor, alimentation, and physical training. There is variation too in the conditions of discharge and restoration; sometimes it is economically surrendered, at others wastefully lost. Mental or emotional excitement disturbs the equilibrium more than all other causes. This factor differs again in the varying degrees of the normal physiologic irritability of individuals and conditions of the same person.

Mental equipoise is the greatest defence against nervous bankruptcy, so much so that the Greek philosophers gave the palm to equanimity as the fundamental quality of success and human power. As this attitude is assailed the loss is shown in the central nervous reservoir, the store-house of energy and power. If life is lived under a continuity of worrisome fatigations the loss will tend gradually to overbalance the storage of force and the tissues are more than tired: they become exhausted—structurally as well as kinetically depleted. Hence adequate time is absolutely necessary for repair of tissue as well as for the restoration of functional capacities.

It is somewhat otherwise in the restoration of tissues exhausted by acute disease only. Here the reserves of fat, of muscle tissue, and many other more or less unstable cells, are reduced in bulk, in proportion to their instability. The integrity of the organic structures remaining unimpaired, only cell depletion being sustained, and the kinetic coefficient still intact, the question of restoration is chiefly one of alimentation. Little more is needed than time and rest proportional to the period of the progress of the acute and rapidly acting disease processes.

These and correlated facts must not be lost sight of in approaching a case of mingled bodily and mental exhaustion, in which is included the factor of depletion of that complex entity called nervous energy. The first step, then, is to overcome those states resulting from lowered nutrition of cells, neuronic and other. For this the systematic use of Mitchell's recommendations are widely employed by physicians in private work, and more largely in the increasing number of well equipped sanitariums. The results obtained are often disappointing, rarely as to the immediate effects, but chiefly in the matter of permanency. Some physicians get results that are lasting, wholly, or partly, satisfactory. Why others do not, with practically the same materials, warrants investigation.

This paper will be divided into, first, an inquiry into the indications for treatment by rest and isolation, the difficulties encountered, and how these are to be met; and second, a description of the procedures to be followed, and the precautions to be observed. Before it is determined to undertake the time-consuming series of measures known by the term "rest treatment," one must always bear in mind that, as has been clearly enunciated by Mitchell and his followers, all other reasonable measures must first be faithfully tried.

The conditions for which rest treatment is most commonly suggested are, first neurasthenia; and second, a complex disorder called hysteria. On either side of these lies a state known as "general nervousness," a term vaguely used and imperfectly understood by both the laity and the profession. The causes which conspire to bring about the attitude of the organism (for it can more accurately be stated than described) are numerous always and similar. While it is possible for a thoroughly healthy person, young or old, to fall a victim to this train of symptoms, it does not often happen. Far more frequently can there be demonstrated a neuropathic heredity; or again, education and environment join in establishing a neurotic quality upon which exciting causes act more or less readily. In this category must be considered that variety of physiologic irritability which follows in susceptible underdeveloped folk, on prolonged or exhausting disease, or upon anatomic defects, such as refractive errors of the eye, malformations or lack of development of the genital organs, the naso-pharynx, the respiratory organs, and the digestive tract including the teeth. Among these factors exciting causes, of less or more potency, find a well prepared ground. And first is objectionable early training, repression of normal spontaneity, or lack of opportunity for, or encouragement in, healthy animal propensities. Be it understood that even a distinctly neurotic child reared by wise hirelings under liberal opportunities may develop splendid qualities. Much might be said in favor of the prophylaxis of nervous and mental diseases taking as a text the relationship of the child to neurotic parents.[1]

Pages might be written upon the contributory causes which are potent upon an organism which lacks, in some degree, nervous equilibrium. Certain of these causes, if severe enough, or complex or prolonged sufficiently, are adequate to produce disturbances of great severity upon a constitution of unimpeachable vigor.

INDICATIONS FOR THE REST TREATMENT

It would seem at first sight that there should be no difficulty in determining whether a person showing evidences of exhaustion by reason of the harassing vicissitudes of life, or protracted remissions in stages of ill health should need treatment by rest. But it is by no means easy. A specially trained observation or possibly an instinctive power is required to determine whether it is best to pursue the full measures of isolation and rest. The element of forced feeding, a third factor always to be reckoned with, again demands much judgment. Those observers among clinicians of proven ability who declare that the rest treatment is of little value, and indeed, some go so far as to say, productive of harm, have made these mistakes. Again, it is often a fact that from lack of experience or confidence in the final effects of the measure, these same physicians may not have persisted sufficiently long, nor have known how best to modify the early restlessness which is often shown by the patients subjected to an onerous and protracted series of measures. Again, it is true that as a rule men do not endure treatment by rest so well as women; although, it often happens that they need it even more. We will assume then, in judging of a patient, that all reasonable measures calculated to relieve the various organic disturbances have been employed. When there is only a moderate degree of exhaustion, that which Dr. Mitchell calls a treatment by partial rest may suffice. This consists of regulating the day systematically with a proper alternation of moderate activities and rest lying down, with outings, and tonic measures. The cardinal principle here is, as in the full rest treatment, a complete and dogmatic systematization of all the waking hours. It is scarcely necessary to detail these measures which must necessarily vary with the needs and capacities of each one. If this combination of regulation of diet, times of eating, passive and increasingly active exercises, with possibly hydrotherapy and static electricity, fails to make the person gain, then the question of absolute rest and isolation must be confronted. The first desideratum is that the physician must be prepared to study the individual from the standpoint of temperament, previous training, tastes, prejudices, and capacity for obedience. He must continue this supervision as conscientiously as though the one patient were occupying his individual attention. If his time is so busily occupied that he cannot do this, then he must act through qualified assistants who must watch all the indications and changes and report to him the essential points daily. There are many conditions fairly simple and easily to be understood for which rest treatment will prove of the utmost advantage, even if it were merely for the nutritive repair and rejuvenation of the exhausted centers of innervation. These, there should be no difficulty in recognizing nor in treating. The psychic element in them may be free from special obscurities, volition and control may be maintained; in short, good sense and a reasonably cheerful submission and an intelligent co-operation mark these persons. These are the patients which a physician not especially qualified by wide experience will find small difficulty in treating, and with good success. Of all the cases subjected to this treatment that I have been able to watch in the practice of others as well as in my own, this class of sensible folk is in large excess. The really difficult problems, where success can only come through the exercise of the keenest insight and the most accurate estimate of their needs and possibilities, are encountered in those cases compounded of will loss, deep-seated prejudices, feeble yet annoying obstinacies and marred dispositions—which have been portrayed by me in the article on the Repair of Will Loss (op. cit.).

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[1] The Nervous Mother, By the author. University Med. Mag., July, 1895. Neurasthenia in Children. International Med. Mag., Dec., 1902.

A brief summary of these conditions will be presented here in order to make clear what follows. A large number of those who apply for advice to the physician, have suffered less or more for months and years; sometimes actually ill, again improved but not well, all the while their attention is becoming more and more focused upon themselves. These sufferers from the common sequence of long-continued illnesses and half-invalid states will baffle the best efforts of almost any physician, and they form the ground from which much future wretchedness may and does arise. They gradually drift into one or another phase of self-deception and mental misdirection until not only are invalid habits contracted, most difficult of dislodgement, but finally there comes a total incapacity honestly to relate symptoms or to judge of the due importance of one or another item of indisposition. This causes the sufferers unwittingly to deceive themselves and their medical director. Hence two points are ever uppermost: first, the varied parentage of maladies composed of divers factors, both physical and mental; and, second, an impaired capacity to judge of these, or any matters, and to narrate them honestly. The springs of action by reason of frequent interferences discharge irregularly, and control-influences, intellectual and volitional, become palsied or almost dead. One feature is common to all such cases—the consciousness becomes distorted, deficient in normal volition or impulse, amounting to what I call, for want of a better term, Will loss, and upon this single factor must our attention be centered.

These patients with will loss become oftentimes exceedingly accomplished critics of medical men, their manners, methods, powers, limitations, and above all their peculiarities and weaknesses. Thus the latest physician consulted is in a false and perilous position, and woe betide him if he fail to realize his jeopardy. If the patient applies for advice frankly and puts himself, or is put by his family, unreservedly in the hands of the leech selected, all will as a rule go well.

Any sensible, competent medical man who once becomes familiar with the problem and gets the patient fairly in his control is probably able to arrange a line of procedures; and, if he be resolute and resourceful, can push these to a successful issue. Far otherwise is it when a perpetual distrust befogs the already sick consciousness—when suspicion, distrust, and conceit induce the sufferer to begin treatment with all sorts of reservations, stipulations for special privileges, and in various ways to assume an attitude of unhelpfulness, advancing weird theories on the "case," and specifying the particular atmosphere in which alone such a one is content to dwell while submitting to repair measures.

Here are all the elements of difficulty and disaster, especially when these reservations include submission to only so much of treatment as is practically unessential, excluding too often those points absolutely imperative. The first prerequisite of relief is a clear understanding of the pathologic items; next come the individual peculiarities; then the family views—including the amount of authority with which they are willing to invest the physician, who should have full control. The financial status also must be defined—stating how much is possible to be done in the way of furnishing rest, nursing, change of scene, etc. Then comes a clearing away of all old moth-eaten relics of medical misconceptions. A healthy mental attitude must be induced, at the head of which stands the promise of a fair-minded obedience to that mutual agreement which should be defined and entered upon betwixt patient and physician. Then follows a host of needful points, chiefly of a psychologic nature, involving moral problems; and finally, a gradual upbuilding of wholesome volition, an acquirement (in which the physician acts as "guide, philosopher, and friend") of healthy views on the duties and privileges of life as it opens up to each one.

THE PROCEDURES OF THE REST TREATMENT

Before outlining the measures employed in the full treatment by rest, isolation, and forced feeding, let us discuss the results to be expected. The primitive conception is to secure relief from pure weariness grown so great as to warrant or demand a long period of retirement and recuperation. This sort of disability needs no special skill or array of determinate equipment. The family doctor is abundantly able to do all that one so circumstanced needs.

Sometimes it is possible to use the home, but it is always much better to place the patient anywhere else, because no one can withdraw his mind from attention, conscious or unconscious, to familiar sounds and movements which will carry an exaggerated significance to a patient in his own home. Nothing is more fatiguing than to hold the attention on matters of routine responsibility when nervously overwrought, hence absence from home is of paramount importance in every case of great exhaustion. But great exhaustion, from whatsoever cause, becomes a disorder or disease of the nerves. Every case of nervous disease must be viewed from these aspects: it may be functional, it may be organic, or it may be and usually is a combination of both. It is rarely enough the one or the other. There is commonly superadded to this another factor: disordered mental states—psychoses, which have been discussed already. Again, the organic disturbances are usually complex, by no means confined to the neurotic phenomena, but evidences will be found of various forms of toxemias and their effects on the vital organs. A period of rest with leisure on the part of the patient and the physician affords a splendid opportunity for the study and repair of recognizable disease. When in doubt as to the residue, the rest and careful regulation of the feeding will aid further a rejuvenation from disabilities only foreshadowed.

The first effect then to be expected from this somewhat elaborate series of measures is to be had from the complete immobility of the voluntary muscles. This point need not be elaborated, because it is a fact familiar to all medical men that relief from even the moderate physical strain of sitting up and occasionally moving from place to place is of large importance. This is pre-eminently true of injuries, as for instance in a case of fracture, although it is not always imperative to employ mechanical immobilization—merely passive conditions may suffice. Again it is recognized to be of imperative value in the treatment of acute fevers, for instance, in typhoid fever, in which every atom of strength which can be conserved should be, to enable the organization to pass economically through the critical periods of exhaustion brought about by the destructive processes of the fever. It should be plain that a period of absolute rest, lying down, with no responsibilities (except the exhibition of obedience) should enormously conserve such forces as exist. Incidentally it may be remarked that the habits of the mind offer a difficulty here, which is oftentimes difficult to meet. After two or three days lying prone, a person of energetic temperament becomes impelled to use his motor machinery, driven to this by the habits of activity acquired through a lifetime. It is a well proved fact that if patients can be kept in bed for between one and two weeks, the relaxation is so complete that they no longer desire to get up and will as a rule remain contentedly thus for as long as it may be necessary to keep them. And finally there is frequently great difficulty in inducing them to get up and move about in the carefully regulated fashion which must be employed when the time comes and the conditions warrant this variety of physical trainings. This point will be enlarged upon later; the steps of progression will be outlined, and the degrees of activity which should be most carefully regulated will be clearly set forth.

The next point of value which we strive for is the isolation. As has been remarked, much fatigue results from a continuity of attention upon ordinary affairs of life and much more when responsibilities are added. That a patient in these conditions should see no one but those concerned in carrying out the measures is not so obvious a necessity, but nevertheless it is paramount. After a time it may be admissible to see some members of the family, but this must be long deferred. A more difficult point to make clear to the patient is the need for holding no communication with family or affairs. Messages except of the simplest character, letters, telegrams, and even the daily papers, are hurtful in varying degrees. The customary rule is to omit entirely all such communication with the outside world until in the judgment of the physician it is admissible. Then, or before if it be absolutely necessary, the nurse is permitted to write a few letters at dictation, then little by little the news of the day, carefully selected and censorized, may be received.

In some cases early, and in others late, the nurse is allowed to read from some interesting book, for a specified time, once or twice a day. The character of this reading must be carefully selected. The best thing is the simple mental pabulum of childhood, of which the beautiful classical fairy tales and ancient romances are the best. Many a patient has expressed to me the greatest satisfaction at the opportunity offered of renewing their acquaintance with these delightful Old World romances, which may have come to their attention in early life, or, as often happens, appear as a revelation of things long missed. Indeed, it is a matter of much moment for the physician to be sufficiently familiar with the best of this class of literature, both ancient and modern, which is usually that selected for the budding intelligence of youth, and by means of which much beautiful and admirable mental, and indeed moral and philosophic, culture has been propagated from time immemorial.

One is almost tempted to turn aside and discuss for a moment this subject of literature suitable to offer under these conditions. It would be a useful essay for a master of letters to write. This would be equally valuable for that larger audience encountered by all medical practitioners among convalescents. It is also a fact, not so well known but of the highest importance, that in the mental and character growth of every one, an essential element of full mental and character growth can only come through the enjoyment of seasons of solitude and opportunity for quiet reflective thought. In the lives of all great thinkers, who have swayed the world from their intellectual eminence, it will be found that once or oftener they have lived for long periods separated from the busy routine of affairs and have been provided with, and accepted, opportunity for solitary communion with themselves and good books, through which is acquired the basis of right thinking. Next, it will be seen that this is not a period of punishment, nor even of banishment to be patiently endured, but rather a welcome occasion for acquiring conditions of mind of which all of us have need and few get enough; thus can be secured a moral perspective of one's self. Perhaps when all is said, this is the largest factor for good obtainable in the rest treatment.

Next comes the question of nutritional repair. We nearly always find digestive disorders a prominent feature in those who come to need this treatment. The basis of this is primarily, and sometimes solely, the results of defective innervation. How can digestion, assimilation, and dissimilation proceed normally when the central nervous forces are exhausted? It has been taught by Mitchell, that nearly all the disturbances of the digestive functions can be brought into line by the restfulness thus engendered. Indeed, most of them can be; certainly the more prevalent ones. There will be found at times, however, a disturbance so deeply seated, or it may be a lesion in the digestive organs, that may require especial attention. Sometimes the impairment of digestion will not thus come right by the rest alone and specific help is needed. If the physician be not thoroughly expert in this department he will do well to seek in consultation some one who has given undivided attention as a specialist to the digestive tract. It is well to know the precise status of the various organs, to have them formulated, and, if any one be seriously disturbed, to direct particular measures to this end. Sometimes there will be found so marked a degree of impairment of the digestive organs that atrophy of the peptic glands with absence of the normal secretions, especially hydrochloric acid, has occurred; and this though often thought to be hopeless, nevertheless, is capable of repair. More often there are disturbances of motility, eructations of gas, water, brash, sourness of the stomach, fermentation—stomachic or intestinal, and delayed digestion. The quickest way to remedy this is by gastric lavage, which serves the double purpose of cleansing the gastric mucosa and the duodenum, and above all restoring the motility. Another excellent device is—giving plenty of water—to omit all food for a few days. Sooner or later, after a rest to the digestive organs, the appetite returns.

The bowels are often obstinately constipated and therefore require special training. The convenience of having the patient under entire control enables us to use measures which might otherwise never be properly pursued. The occasional use of high enemas of alternating stronger and weaker solutions of salt followed in some instances by direct massage over the intestines serves several purposes of cleansing and of peristaltic stimulation; and as much of the saline solution as is absorbed frees the surrounding lymph channels from toxins. By these means I have occasionally cured completely a lifelong habit of constipation. Enemas must not be employed oftener than once every few days, otherwise the muscular tone of the intestinal walls may suffer impairment. Careful attention to the centers of arterial supply which can be reached in the lumbar regions by special mechanical excitation and also, when there is a tendency to retention in the gall-bladder, by a direct manipulation of this near the border of the ribs, combined with pressure on the upper lumbar segments, will go far toward bringing about a cure. This had best be understood and practised by the physician himself, because the average masseur cannot be trusted with special manipulations which are pre-eminently the province of the physician.

THE GENERAL CONDUCT OF THE REST TREATMENT

The various factors or procedures involved in the treatment by rest, isolation, and forced feeding may be outlined as follows—assuming that we have a case in which the most complete details of the treatment are to be followed out:

Rest.—The patient is put to bed and every influence used to induce as complete surrender as possible. This full relaxation is the basis upon which the other measures are built. It will be found that many persons at once welcome the perfect freedom from exertion and external disturbances. Others chafe under this until they become inured to it, which comes about usually in a week or ten days. It is often necessary for the physician to confine his conversations with the patient for this length of time to presenting in a variety of ways the value of this element of quiet and repose with hints or even orders how this shall be effected. The overtense person finds it difficult to let go and allow the body to be entirely supported by the four stout legs of the bed; she rather holds the body in various constrained attitudes involving unconscious muscular action. In securing complete tissue slackening an important auxiliary is a wise masseuse. It was always our custom in training the classes in massage at the Orthopedic Hospital, where, for sixteen years, I taught the essential principles and the physiologic reasons of the art, to spend some time emphasizing the necessity of securing a complete letting go of self by the patient. This is done by a little explanation and repeated demonstrations. A limb which is about to be rubbed is held in the operator's hands in such a way as to allow, in fact compel, the abandonment of all voluntary motor action; otherwise the tissues cannot be reached and manipulated satisfactorily. As little as possible of speech should be encouraged until later in the treatment when judicious conversation is not amiss and possibly of value, provided it be confined to certain subjects or methods of presentation in which it is most important for the operator to become skilled. In very few instances does the patient fail to become, in due time, entirely malleable in the hands of the operator.

Outline of the Daily Routine.—Mere routine has its value provided it be entirely passive—involving no effort on the part of the patient. When complete relaxation has become established and isolation recognized as inevitable, the subject looks forward with some little healthy interest to the rotation of the day's doings. I have again and again heard the remark from patients, that instead of this being a period of rest, they never were more busy in their lives. As a matter of fact the mind views external things differently, provided the standpoint is altered. When I meet such a remark by the query whether the routine is agreeable or otherwise, it is common to find it admitted that the procedures always raise a little curious expectancy and the various measures are anticipated with steadily increasing interest. The difference in the mental outlook is simply this: during the period of activities by which exhaustion was produced, there was a constant succession of events in which the patient exercised a more or less constant effort; the attention was perpetually on the strain, as is proper and healthy enough if the organism be in good equipoise, but becomes fatiguing when the driving forces are lowered below normal. One of the most valuable working formulas is that axiom of Benjamin Franklin's in which "Poor Richard" states the important rule: Drive your business, but don't let your business drive you. The patient who requires the rest treatment is no longer in the position to drive his or her business, whatever it may be, but has fallen into the condition of being unable to do so and therefore the business drives him. A further analogy that I often use is that of a ship: so long as the ship is under way and proceeding by its ordinary motor forces it can be steered satisfactorily. If the driving forces become too strong so that the capacity for guidance is lost in the overwhelming strength of the elements the ship can no longer be accurately directed and becomes buffeted and in danger from this misdirection. Again, unless the various measures follow in regular progression the same results cannot be expected, because, if for no other reason, it is an expensive process, on account of the time lost.

The best results are had from modelling the day much after that of an ordinary working person. Measures should begin early in the morning. The patient usually needs to be aroused at about seven o'clock, or even earlier in summer weather. With the first peep of dawn if the patient is awake, or by seven o'clock at the latest, when if asleep the patient should be awakened, the nurse should bring some hot drink to the bedside, and administer it. A small cup of cocoa is in some respects the best, or it may be hot milk or weak coffee or tea. Cocoa is useful for those who need to be fattened; the tea perhaps better for those who are already burdened with soft flesh. The hot drink acts both as a food, enabling the patient to bear the fatigues of the subsequent bathing and partial dressing, and also, on account of the heat, as an admirable heart stimulant. Indeed, a cup of hot water in the morning is a far more competent eye-opener than any combination of alcohol. This can be proven beyond peradventure in the case of men accustomed to take much tipple who often became more enamoured of a cup of hot water, with possibly a slice of lemon in it, than of any concoction emanating from a bar.

After the cup of hot fluid, the nurse administers a brisk dry rub or a bath. The sponge bath is best as cool as possible; to those who can endure it, it should be cold. It also is the better for containing some stimulating property, as salt—a tablespoonful of salt in a basinful of water. The patient lying between blankets to prevent all chill, the nurse takes one limb at a time, bathes it gently, and afterward rubs the skin to a good pink glow. As each part is disposed of it is carefully protected by the blanket and the next approached. This occupies probably half an hour; then a little rearrangement of the clothing and the subject is made comfortable for the day.

About eight o'clock comes an ample breakfast, which will be found in no way interfered with by the small cup of drink earlier. After the breakfast a period of an hour's rest follows while the nurse attends to duties elsewhere, and the patient is not allowed to be disturbed in any way. The second meal is given a little after midday, about one o'clock, and takes the form of a liberal dinner. At six o'clock comes the last meal, a fairly substantial supper. After each meal a period of an hour should elapse in which the patient is to be kept entirely quiet. Between breakfast and dinner, about half-past ten, fluid food is again given—milk, broth, or soup. In some cases this is repeated between dinner and supper; and at nine o'clock, or before the final tucking in for the night, it is customary to give again a glass of milk, preferably warmed.

The massage should come either in the morning, just before the milk, or in the afternoon, midway between the meals; the electricity in that half of the day not occupied by massage, either forenoon or afternoon. On going to bed at night there is frequently given a dry rub to the skin with a coarse towel, or, if salt is not used in the water in the morning bath, with what is called a salt towel. This consists of a coarse linen cloth dipped in a strong salt solution and hung up to dry; it makes a pleasant stimulating application to a flabby skin. Massage is best used in the morning, and is usually followed by the glass of milk already described, the patient again lying entirely quiet for an hour. Electricity is best given in the afternoon, and need not be followed by anything except perhaps a little cheerful conversation. Indeed, the physician's visit is most acceptable during the latter half of the day when the occupations are somewhat less systematic and the diurnal ebb and flow of vital forces are at their lowest. The patient is then most lonesome, the thoughts dwell upon home and other emotional matters, so that the visit of the physician is welcomed in turning aside these discomforting ideas. The electricity need not occupy more than half an hour—making an agreeable break in the day's work. This may be administered by a young physician trained for the purpose, or, in some instances, by the nurse; but it is of distinct value oftentimes to have a variety of personalities in these routine measures.

The daily evacuations should be sedulously regulated. It is well to follow the habits of each one as to the customary time of the bowel movement. If this can be secured in the afternoon, it is sometimes better. However, a full daily evacuation of the bowels should take place at some regular time, and the will of the individual should be trained to bring this inevitably to pass.

Not the least factor of value in this whole episode is the establishment of a thorough systematic habit of body and mind. At first there should be entire dependence of the patient upon the attendants; later, as active measures are instituted, the patient should be taught and urged to direct her own volition in compassing the spirit of the various measures which she must soon or late depend upon herself to continue.

The urine must be looked to, its quality and quantity carefully noted; fixed times for the voidance of the urine are very useful.

The temperature should be taken regularly, at least twice daily, to observe the fluctuations and unknown tendencies. It will often be found about 100° F. for two or three days; then it may drop to subnormal for a few days more, and finally become and remain normal.

Diet.—An ordinary, liberal, wholesome diet, not especially limited in any direction, is, as a rule, permitted and encouraged. If there be manifest digestive difficulties, they are to be met by such measures as may be plainly indicated. When a marked loss of digestive tone exists, it may be necessary to begin with a small amount of peptonized milk every two hours, rapidly increasing the amount and adding various semi-solid and solid articles. If the patient be profoundly anemic, as often happens (and this may be accurately determined by a blood estimation), forced feeding, by which is meant urging food upon the stomach, is early instituted. When the patient is exceedingly feeble, the nurse is directed to feed with her own hand, taking up the food bit by bit and handing it to the patient on a fork; also cajoling her to drink the necessary amount of fluid food to the last drop. Many times a person is indisposed to eat from sheer fatigue in handling the fork. This may seem doubtful, but is nevertheless frequently demonstrated. A person who has for months or years eaten, even as the birds eat or are supposed to eat, little or nothing, can thus be induced in a very short time to take such an enormous quantity of food that no one is more surprised thereat than herself. Not only so, but folk whose digestive capacity has been considered the most diminutive can soon swallow and assimilate astonishing quantities of provender. Milk should always be used as a faithful ally. Of course, a large number of people have come to believe that they cannot take this important article of diet without distress. Like other self-deceptions, this is usually provable to be a myth. Of the one person out of five who declares she cannot take milk, it is fair to assume that only one in thirty or forty can really not do so. This milk—sound, wholesome cow's milk—should be given in its full strength. Many times it may be necessary to reduce this by skimming or by dilution. Sterilization, as is well known, impairs the nutritive value of the milk. Very rarely it is impossible to secure thoroughly good milk; it should be as fresh as possible, be prepared rightly at the dairy, and it should come straight to the patient, twice a day. The dairyman is not so dishonest a fellow, as he is often regarded. When there is loss of appetite or marked digestive disturbances, it is often of value, at the beginning of the treatment, to omit all food for several days—until the appetite returns. Forced feeding has its perils and often its grave discomforts, which judicious water-feeding (without nutriment) will largely correct.

A considerable portion of those for whom rest treatment is needed are fat, anemic women. In the management of these cases much trouble arises. For them it has been found best to use a skimmed-milk diet, somewhat after the manner of Karell, of St. Petersburg. This, in brief, consists of specified amounts of milk, say about four ounces every two hours, and nothing else except water allowed. If hunger grows distressing, this amount may be rapidly increased half an ounce at a time each day until eight ounces are taken every two hours, then ten or twelve ounces every three hours, and finally, in a short time, small amounts of meats, fowl, oysters, etc., with a little toasted bread, a few stewed prunes, etc., until the diet is gradually increased, and sufficiently. Upon this diet the fat soon begins to melt away, and in a few months' time, along with powerful massage, sometimes given twice or even three times a day, the whole contour is changed, not only architecturally, but mentally, and the characteristic, peevish whine of such women is agreeably modified. Thus in two or three months they can be literally made over upon a much improved plan.

Peptonization is at times employed for milk, when the difficulties of digestion are pronounced, and this, to my mind, is best done by what is known as the cold process. About three grains (0.2) of pancreatic extract and five grains (0.3) of sodium bicarbonate are put into a glass and rubbed thoroughly with a tablespoonful of milk. To this is added the rest of a glassful of milk. The mixture is then agitated in what is known as a shaker, or tin cup placed over the top of the glass, and administered immediately. Not only is the milk thus rendered free from latent acidity, but agreeable—the mechanical addition of the air giving it a lively, bubbling quality. Ordinarily peptonization is not necessary. In extreme cases it may be carried on by subsequent warming for twenty minutes or a half hour, but this gives the milk a disagreeable taste, offensive to most persons. Various devices are often needed to make milk acceptable. One of the best is to add a little burnt sugar which kills the "cow taste." Another is to add a sparkling water, a pinch of salt, various flavorings (of which maraschino cordial is one of the best), a small amount of rum or brandy, etc.

Soups are used largely as an auxiliary food, and good broths, especially chicken and oyster broth. Clam broth, when procurable, is a useful addition to the dietary, especially if the patient's appetite is not good; in the second or third week it may be substituted with advantage for the milk between meals or at night. As to the articles which constitute the regular meals little need be said, except that they should be well prepared, savory, well served, and free from objectionable qualities. It is a matter of common experience that a notable dyspeptic will, after a few weeks of this treatment, comfortably take a breakfast consisting of what are supposed to be very indigestible substances, such as buckwheat cakes and sausage, with perfect impunity, or even that monumental rock on which so many split, Boston baked beans. A lady said to me on one occasion: " They gave me beans for breakfast, great fat beans, with brown coats on them—regular rubber overcoats. I ate them because you said I should take everything offered and to my unspeakable astonishment they consorted most happily with my tissues."

There are many details to this matter of diet of which it might be interesting to speak, but confining myself to the essential matters only, little more need be said. The habit of eating slowly is given an admirable opportunity of establishment—a precious possession. If the act of feeding be tiresome or the patient show signs of slighting this function, the nurse is instructed to feed urgently, almost forcibly, always with kindly tact and encouragement. Thus twice as much is often disposed of, and without any subsequent discomfort.

When fattening is desired much butter is used, always the best, spread as liberally as possible on the bread or toast, as much as six ounces a day, as von Noorden teaches.

MEDICINES.—One of the chief advantages of this plan of treatment is the absence of drugging and the dependence upon the rational tonics of rest, system, food, passive exercise, and freedom from artificial stimuli and other forcing agents. It is important to keep the bowels active, so that assimilation may be encouraged. The medicines most important are those which aid digestion, that is, the nutritive tonics, such as malt, which does more than aid in the digestion of starches, and seems to exert some curious effect beyond this function. The form of malt may be the fluid extracts, which are usually little more than a bitter, slightly alcoholic stimulant, with small diastatic power. Some of the preparations of this upon the market are utterly valueless; those that seem to be useful very often soon deteriorate as they become popular. For my own part, I rely largely, when needing this particular form of tonic, upon the reliable Guinness' stout, which is a little high in alcoholic strength, but far more agreeable to the taste. The Bass Company makes a good malt, and there are many excellent American malts and more of worthless ones. The sugary extracts are much alike, and sometimes do better than the fluid preparations, especially when it is convenient to combine these with cod-liver oil, or the digestive ferments, such as pancreatin. These are best administered with the meal. At times the Guinness' stout is a very valuable aid to appetite, taken just before eating. Cod-liver oil is generally needed, and can be given in many ways, but perhaps the best is an emulsion freshly made by a reliable chemist. Capsules are convenient and best for those who are distressed by the taste or after-taste. Iron is the great sheet-anchor, and should be increased from a small dose until large amounts are taken. To those with whom iron mentally disagrees, the pyrophosphate or some such tasteless preparation, is given in the malt. As the patient begins to be more active the lactate of iron in a pill with strychnin may be used. For obstinately slow bowels a small amount of aloes may be added to the pill, gently to stimulate. Sometimes it is important to help sleep in those who have been much disturbed in their rest, and for this the simpler hypnotics are employed for a short time. When tranquillity is secured all such are promptly withdrawn. The mere habit of sleeping often becomes improved by the other measures employed, and artificial aids are rarely needed long.

Massage and Remedial Movements.—The enforced inaction continued through many weeks would produce objectionable effects unless something definite were done to rob rest of certain evils. The most important agency for this is to employ regularly a generalized massage or manipulation of the muscles and tissues by which they can be maintained in a healthy condition until the time arises when active muscular movements can be resumed. If this were not done as a rule there would be too great a gain in flesh, chiefly fat, but all the cellular accumulation would be loosely organized and unstable. An index of the good which the rest treatment is accomplishing is a normal progress in the accumulation of weight which should be not too swift. It is an axiom that poor massage is better than none at all, but by this is meant lack of skill in the manipulations failing to bring about the best results; it does not mean over zealous or forceful manipulations, which may do, and sometimes result in, much harm. A fair index of the value of the massage is the equation between the amount of food taken and the degree and quality of the digestion. It should produce a proper amount of tissue change and waste by which the solidity of the tissues is improved. It is surprising that certain physicians, especially neurologists, assert that they find massage of little value and are prepared oftentimes to omit it during the progress of rest treatment. In my opinion it is impossible to get along without it if satisfactory results are expected. The difficulty is that so few physicians are alive to the fact that in mechanical therapeutics we have an agency of the most invaluable sort but of which they personally know almost nothing; nor will they take the trouble to inform themselves on the subject, preferring to delegate it to another person whom they regard in the nature of an inferior. The time must come and that speedily when physicians will realize that one of the largest factors in therapeutics is mechanical excitation of the nervous control mechanism. By skilful manipulations the vasomotor system is capable of being influenced and through this a large part of the organic activities.

It may be we shall always employ for the routine procedure of passive exercise a special expert in massage and remedial movements; but in order that we may get the best results, the medical adviser must himself clearly understand his physiology, the enormous possibilities of mechanical excitation of centers, and he must himself perform certain parts of this labor. It is impossible, in the brief space permitted for this article, to go into my own convictions on this subject which I shall do elsewhere. I can only here emphasize the fact that physicians will do well to keep abreast of the progress in mechanical therapeutics, so that they may at least do what is done in those countries where the subject is adequately taught and be able to direct intelligently the work of the experts. In Sweden, Norway, Denmark, and Germany, the courses of instruction occupy from one to two years, and the physicians themselves also make a study of this art. They are enabled then intelligently to direct. This they do easily by a carefully written prescription varying with the needs and conditions of the patient, specifying what shall be done from time to time.

In this country the courses in massage are seldom more than three, sometimes as much as six, months. The character of the work thus done is unfortunately superficial. A mere handful of physicians know anything about the subject at all and hence their directions are of small value. The temptation is for the skilled European masseurs to proceed upon their own initiative which brings out, too often, evidences of arrogance which their superior knowledge warrants and hence there is likely to be a collision between the medical adviser and the masseur who thus interferes too much in the conduct of the case. The consequence of this is that the simplest thing for the physician to do is to omit the use of an agency which he cannot properly direct and to belittle its value. It is well to bear in mind, also, that for very feeble patients massage is capable of producing fatiguing effects of a somewhat serious nature, even if the methods employed are excellent. When it is held in mind that undue pressure upon certain areas produce powerful effects upon arterio-motor innervation the physician at least must be on his guard to warn the operator how to avoid this. Massage as used in the routine fashion of the rest procedures is usually given in the middle of the forenoon, and the electricity in the middle of the afternoon, or this may be reversed if necessary. At first the time occupied should be not over twenty or thirty minutes and be rather a slow gentle effleurage with passive movements of the joints. At first the manipulations should be stroking, then kneading in character, and gradually increased until the upper tissues are made to act upon the lower—until gradually the deepest tissues are reached and pulled and stretched. As the patient becomes accustomed to this, in a week or ten days, deep kneading is used, increasing the force until all the muscle masses are squeezed and relaxed and the long tendons pulled upon and rotated without pain to the patient, bringing about much cellular stimulation. This should always result in a feeling of comfort and satisfaction akin to that which follows active exercise. Massage under these circumstances is chiefly for the purpose of affording a passive form of exercise which the condition of the patient would not warrant, and it is generally accepted that a full hour's massage treatment is equivalent to an active walk of five miles. This brings no strain upon the heart or the central control mechanism and hence does not fatigue either the circulation or the attention. As the treatment progresses the massage proper can be more quickly and forcefully accomplished, and passive movements of the limbs are more thoroughly used until considerable force is exerted on the tendons and ligaments by which the joints both large and small are kept in full elasticity and tone.

Toward the end of regular treatment, it may be six or ten weeks according to the strength of the patient, the resisting movements are practised. By these the entire motor mechanism is brought into play and the physical vigor of the individual is oftentimes made to become not only as good as it was under normal conditions, but trained into a better state than ever before. Then this is supplemented by fuller regulated activities until it is not rare to find that a patient subjected to this routine can be brought up to a degree of physical efficiency which she had never expected to enjoy. Let me say here, speaking from ample experience in all forms of athletics and physical training, that I am certain more and better effects can be wrought upon the bodily powers by these systematized remedial movements, if intelligently directed, and above all if the patient judiciously co-operates, than can be accomplished by any amount of undirected free exercises, no matter what these may be. I have often been able to take persons profoundly invalided, and by rest treatment or its modifications, carry them beyond the point at which they began before falling into the condition of neurasthenia, and at the end of a reasonable period furnish them with a degree of muscular vigor of which they had never believed themselves capable; and all this effected more through the agency of systematized self-directed effort than if they had enjoyed a period of ample leisure with every opportunity to follow out the dictates of their own sweet will, by indoor free gymnastics or in field sports. Not only is this true in the young or middle aged, but it is equally true in those well advanced in years. It is a fact not generally recognized that aged tissues are capable of being repaired to a remarkable degree and improved in their efficiency to the point of regaining extraordinary vigor.

During the progress of the routine massage, certain precautions must always be maintained. The tissues must be taught to become thoroughly relaxed while passive movements are being employed. During the more active processes the very fullest accuracy and force must be elicited in not only the posturing movements but especially when resistance has been reached. The resistance movements are best slow but of increasing force to the point where the movement is completed and thence the limb held in the utmost tension of which the individual is capable for a few seconds. It is necessary to remember that the extensor movements in the arms are more important than the flexor, because most of our common daily acts are flexor movements, and we need to use an excess of extensor movements to acquire muscular balance. It is only through the acquirement of thoroughly well directed and accurate symmetrical muscular actions that our circulatory machinery can be kept at its full efficiency. It is only through these circulatory activities that the nutrition of the entire body can be maintained. It is only through the maintenance of the fullest nutritive interchanges that exhausted centers and nerve structures can be made to regain their tone by which full functionation is acquired of both the body and the processes of the brain.

Again during the application of massage there is relaxation of the superficial capillaries and opening of the sweat glands of the skin. This relaxation is followed by surface moisture and this surface moisture by its evaporation induces latent heat, the escape of which produces surface chill and the ill effects which follow. This must be guarded against by a careful protection of the part after the application of massage. A warm blanket should be used, each part being wrapped up after it has been treated. Some operators use large soft woollen stockings for the legs and the same for the arms. When the procedures are completed these may be removed, and the patient tucked carefully in bed and compelled to lie still and rest for a full hour.

It is an axiom in directing exercises for those not fully strong that a period of rest should supplement that of activity and for the same length of time. When this rule is followed in training athletes it is better, and it is imperative for those who are not in full vigor. To sum up: the procedures of massage, passive exercises, resistance movements and active movements should be systematically increased in proportion to the strength of the individual. It will then be found that surprising results follow until it is quite possible in from two to four months to rehabilitate neurasthenic persons and return them to society not only recovered but strong and more enduring than they ever were before.

Electricity.—As a part of the routine procedures it is customary to employ once a day tonic faradism for the purpose of exciting contractions in muscles which would otherwise not be energized. By this means the muscles are made to contract without the strain of volition and attention. This is a valuable measure, and although sometimes omitted it should not be if the best results are prospected. The form of electricity should be that of slow interruptions secured by means of two electrodes held a few inches apart, on and near motor points for three or four interruptions and first along the body of the muscle and then at each end from origin to insertion. The degree of pressure used should change where the muscle masses are large or small. In this way all the larger muscle masses are made to contract in turn as forcefully as possible without causing pain. In the beginning it is likely to be painful, as is the case of the voluntary use of muscles long out of action, and is sometimes followed by that same soreness which follows unusual exertion. If the patient is agitated at the idea, it is well for the operator to show on his own muscles precisely what is intended and then to detach the cords without the patient's knowledge and go over the body for a day or two or longer until this fear is allayed; then to begin with the very mildest possible currents and gradually increase these as they can be tolerated. Ordinarily it will be possible to use the full strength of a Du Bois-Reymond coil from a single cell. The amount of time occupied is from a half to three-quarters of an hour. The ending is usually to employ the rapid interruption with the negative pole to one foot and the positive pole to the nape of the neck for five minutes each. After this a period of rest is enjoined of an hour. The electricity is best administered by a young physician trained for the purpose. At first he should say little to the patient, but by and by a certain amount of careful conversation is permitted and as the treatment progresses he may make himself interesting and can, if wise, by his conversation and hints largely supplement the efforts of the attending physician. It is very important, however, that he should be equipped with tact and judgment in this particular, otherwise he may do more harm than good and had better remain silent.

The Nurse.—The most important factor beside the wisdom of the physician is the adaptability and intelligence of the nurse. Upon her depends success or failure. She must be the right hand of the physician and work with him and appreciate for him. There must be explained to her precisely what is expected. She must have outlined to her as well as possible the whole status of the case and the peculiarities of the individual. If she is really willing to give her full co-operation the difficulties are then removed one-half. She should be selected by the physician and never by the patient. The nurse that brings the patient from a distance or has been in previous attendance, if a stranger to the physician, is valueless, at least if the case be difficult psychically. No amateur nurse however wise and capable can be depended on. Relatives and friends are useless and worse than useless. The nurse should be a stranger to the patient and if possible have had some experience in such cases. However, a young and intelligent and enthusiastic trained nurse can be taught readily to become invaluable, and the old experienced nurse whose gravity has matured and whose enthusiasms have cooled, is not so good. There is full opportunity here for a woman to exhibit her highest qualities of tact, patience, watchfulness, and character reading. The neurasthenic, or the hysteric, or the hypochondriac, or that complex blend which usually demands this sort of care, will inevitably try the patience of a very angel.

It will sometimes transpire that the nurse must be changed, but this should not be too readily done, because it is a hard matter to judge fairly upon the patient's representations. If, however, the complaints are seemingly well founded this may be necessary. It should not be done too often or worse will follow. It is not necessary that the nurse and the patient have similar tastes and interests; it may be better that they should differ in education or abilities. It may be important to act upon the patient through the nurse by marking out a line of fit conversation which can only be thus carried out and may stimulate to other thoughts and to better conclusions where the psychic element is prominent and the faulty impulses pronounced. The nurse should be warned against discussing symptoms and the wisdom of certain procedures. She must be sympathetic but not sympathize. When the physician makes his visit, it is well for the nurse to make her report before the patient and then to leave the room. Thus the patient may unburden herself of any matters without an audience other than her appointed confessor. The nurse will have her opportunity to say to the physician what is in her mind about the progress of the patient when he has left the room.

In that most important part of the results which are aimed at by the "Rest Treatment," namely, the moral training, the nurse figures largely as a factor. Oftentimes it is necessary to carry out special lines of mental and psychical influence which can be done best by a combination between physician and nurse and whomsoever gives the massage and electricity. Where the psychical conditions are not so important many nurses are capable of giving both of these or one of them, and to receive additional pay therefore. When economy must be practised it is occasionally admissible to have the one nurse in charge of two patients or even three, but this cannot be done unless the extra treatments are administered by other persons and it is never best.

Pages might be written of a most valuable sort on the subject of the relation of nurse and patient especially in conditions such as those under discussion. Nowhere in the whole category of medical or surgical cases are the qualities of a nurse so severely tested. The important points come out more strongly oftentimes during the convalescing period when the nurse and patient are travelling together, as, for instance, to some watering place or in the pursuit of the oftentimes necessary changes of scene and circumstances required to finish and round out the treatment. After the treatment has been pursued it is customary to send the patient with her nurse to some pleasant place in the country or at a quiet sea-side or mountain resort and follow out a species of after-cure. Then the need for the nurse is often greater than before and her qualities put more critically to the touch. If she be well refined she should travel as the companion and is oftentimes not to be recognized as a care-taker. If she have some accomplishments, such as abilities to ride or drive, she is thus of special companionability; familiarity with the various field sports is to be appreciated. One of the best nurses I ever knew, I sent to the country to take charge of a lady who was a thorough all-round sport, and among other things a great horsewoman. I knew what the conditions were and what would be demanded, and it was a most difficult problem to find a woman to fill the requirements. After the patient had gained strength enough to go about she found that the nurse could ride her hunters almost as well as herself and was as capable as she in following the hounds, her favorite amusement and which the circumstances rendered desirable. In other ways this nurse could fit herself into almost any breach and by this versatility she retained her hold over the patient, and only thus.

Again, sometimes nurses accompany patients in foreign countries and for long periods and special qualities are needed here, above all self-reliance and generalship. I have found it necessary to instruct a nurse to exhibit, after a time in a foreign country, a degree of helplessness which compelled the patient to take charge of the travelling and regain her own lost dominance. On another occasion a patient who was becoming very dependent upon her nurse, and who was dependent in all the attitudes of life, found herself in difficulties which proved to be after all most fortunate. The nurse fell seriously ill and the patient had to look out for her, and this solicitude was the means of herself regaining certain qualities which almost nothing else could have effected. In short it will be seen how broad must be the mind, how versatile and capable, to qualify a woman to fill all the requirements of a nurse for patients who are so complex as these. Finally, it is a rule I find important, to change a nurse in about three months. Few can be trusted to remain longer. The reasons for this are manifold as experience will show.

The Convalescence.—To do justice to the subject of the gradual return of patients to greater activities and the final bringing of them back to their ordinary methods of living, would occupy much time. Unless this period be judiciously handled all the good which has been accomplished may be readily lost and the condition of the patient may become worse than before. Something should be said about the difficulties of getting the patient back upon her feet after a long period of rest in bed. This usually requires some encouragement on the part of the physician and sometimes forceful domination. He must be careful to judge accurately and not to be misled by the clever misrepresentations of a hypochondriac who has come to enjoy the rôle of complete dependence. The sitting up should be begun by ticking the time off by the clock, first ten minutes once a day, then ten minutes twice a day, then increasing the periods by five minutes until a couple of hours are reached. Then the little activities should be gradually resumed. Here the nurse must not be too helpful; the patient must begin to use a certain amount of will force. Finally an outing is admissible, a short drive, then a longer; then intervals of walking in the open air, and so on. All this must be accurately outlined by the medical director and the nurse should co-operate in letter and spirit. After the patient has left the immediate care of the physician the nurse must report regularly by letter and if possible the patient had better herself report in person from time to time, at intervals of weeks or months. All these directions seem over elaborate perhaps, unless one has had experience by which to judge of their necessity. Those who have had will readily see how necessary all this is, and unless some experience has been enjoyed by the physician he may make many blunders which will tax his tact and resourcefulness to the uttermost. Indeed, no class of cases are more difficult and in none are the rewards less in proportion to the amount and quality of the work done. If the work be well done it seems so easy to the patient and her friends that it appears to be a matter of course that results should follow. They judge differently when relapses and entanglements arise. They then blame the physician altogether for errors of judgment, and with great liberality. It is an education in diplomacy, and one who has learned to do this sort of work well is worthy of a post of great difficulty in some foreign court, where the highest abilities as a diplomat are required.

One word finally as to the rate of gain. In conditions of protracted exhaustion which is usually accompanied by obvious evidences of nutritional loss, the first direction of gain is in the line of tranquillity, then digestive power, then in flesh and color. These all are evidenced in a few weeks or months at most if all goes well, and no serious organic impairment underlies. The one factor which is most slow to arrive is the capacity for sustained effort. In short it takes a year or two years before a patient with well-pronounced neurasthenia can expect to regain the state of health she enjoyed when she began to run down. This must be made clear to the patient and above all to the family—otherwise discontent is manifest. Indeed, it is a safe rule to promise no full vigor short of twenty-four full months. Anything short of that is clear unexpected gain.

Medicine

THE ETIOLOGY, PREVENTION, AND TREATMENT OF A COMMON COLD

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If any apology is needed for treating of such a common, everyday subject as a cold, I would urge that the more common a disease is the more interesting it becomes when completely understood, and the more often one has a chance of putting one's theory into practice and testing results of treatment, the wider and deeper grows the interest. It may be interesting to record a case of a rare disease, but it is far more interesting to understand completely a common disease which one has daily opportunities of watching.

I am led to write on this subject now by the experience of myself and others who live, and have lived for some years, on what I call the "Uric-Acid-Free Diet." This experience is, that those who on ordinary diet suffered from cold after cold throughout the winter, so as to be but rarely free from it, have gradually come to obtain almost complete freedom from such troublesome, if not dangerous, conditions. My own experience, and I have reason to believe that of scores of others, is that they now rarely or never get colds at all, or that if they are infected it is only for a few hours. In my book on "Uric Acid" (Chapter IX), I have referred to a cold mentioned under the name of the "Ben Nevis Cold" which has this special characteristic: that in those persons who go to the top of the mountain, a cold which they have had at the foot of it takes its departure, and does not spread among those who are with them at the top of the mountain; but these same people, when they descend from the mountain into the plains, not only catch colds and suffer from them, but they catch them more easily and suffer more severely than those who have not been at the top of the mountain. A further instance of the same thing—and we shall see the explanation of both these presently—is that people in training for racing of any kind rarely or never take colds while they are in training, but when they have given up their training they not only take colds, but they take them easily and have them badly.

Another fact of interest is that colds are undoubtedly contagious; at certain seasons of the year they may spread through an entire household, person after person following in regular sequence from the bottom of the house to the top, or *vice versa.* But even in a single household you will probably find instances where a cold has, time after time, not affected one or two particular individuals. The reason why it has not affected these individuals is exactly the same reason that it does not effect those on the top of the mountain, or those who are in good athletic training.

If I had to define a cold shortly, I should say that it is collæmia (that is, excess of uric acid in the blood) plus the local action of cold, plus a microbe. The reason that this cold does not affect certain people in certain bracing climates or in certain conditions of training is that these people have not got the collæmia on which the cold and the microbe can act.

But just as a bracing climate or athletic training protects an individual from cold by preventing collæmia, so will any condition which precipitates or insures collæmia insure also that the individual thus rendered collæmic will take cold whenever exposed to cold or infection. Those in training or those on the mountain get severe collæmia when they alter their condition, and this is the result of the uric acid stored in the body during training or during the bracing action of the mountain climate. When the bracing action comes to an end, they not only suffer from cold, but they suffer severely in proportion to the amount of uric acid previously stored. Precisely similar considerations explain the well-known fact that women nearly always catch a cold which is going through the house at the time of their catamenial period. If they escape it at other times, they almost never escape it then. The explanation is that the latter part of the period and the few days that follow it are accompanied, as I have shown in my book on "Uric Acid" (Chapter IV), by marked collæmia. (See temperature chart, showing that collæmia produces a subnormal temperature just at this time.) Hence, as I have also pointed out, the headache, the epilepsy, the depression and suicide, the bilious attack, or the dyspepsia which invariably in

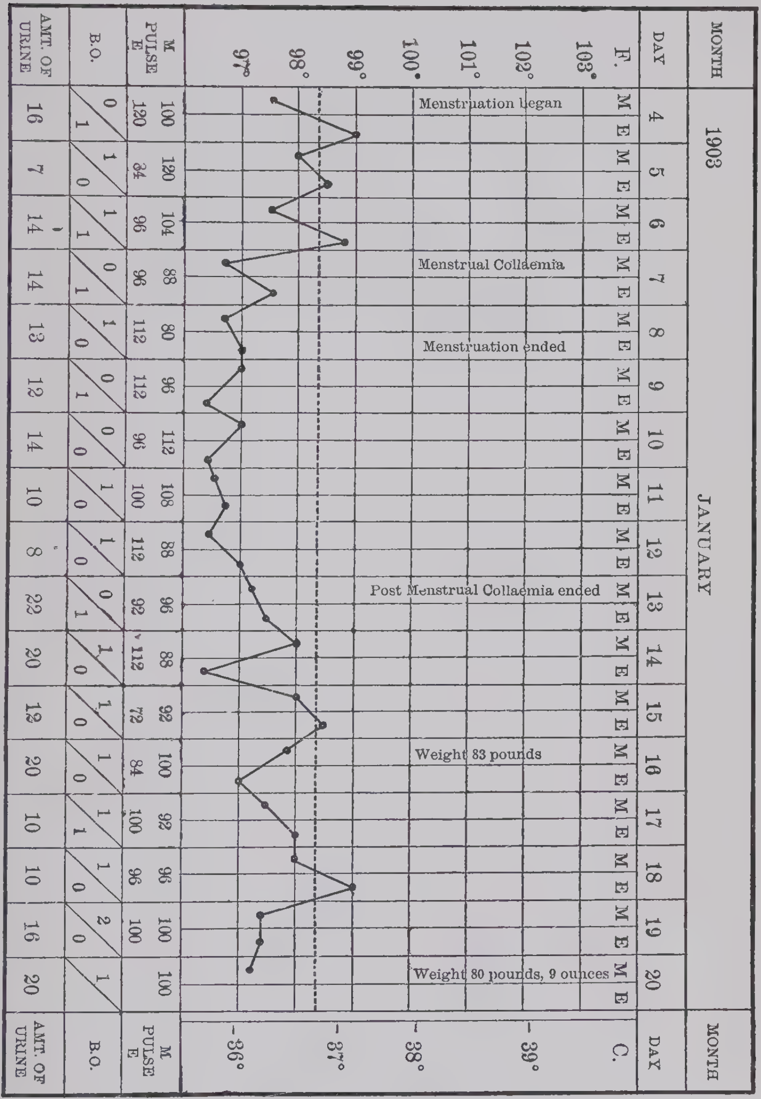

FIG. 1.—Chart of a patient suffering from gastric disorder to show the effects of menstrual and post-menstrual collaemia on the temperature. With this, defective combustion and increased liability to take cold are associated.

women occur during this part of their monthly cycle. They are then in exactly the reverse condition from those in a bracing climate or in athletic training. Collæmia is absent in the one case, present in the other; hence the different reaction to cold and infection.

And this same fact explains the immunity to cold which a uric-acid-free diet eventually gives, for those who have lived 18 months or two years on such a diet rarely or never, unless they take too much food, have an excess of uric acid in the blood on which cold and the microbe can act; hence these factors are powerless for harm. The action of these factors upon one another is, I think, as follows: Given an excess of uric acid in the blood—the effect of a little local cold (such as follows exposure to a draught, or the absence of a scarf round the neck, or having one's hair cut too short, or some similar apparently trivial exposure to cold) is that some of the uric acid from the blood is precipitated locally upon the part affected by the cold, and this causes a local irritation, in fact, a gout of the part, and this local irritation forms a most favorable soil for the microbe, and the first microbe that comes along settles upon it and thrives and flourishes enormously. Then the microbe increases the local irritation, and the increased local irritation leads to the ever-increasing precipitation of uric acid *as long as there is plenty of uric acid in the blood to be precipitated.* Thus the cold and the microbe work hand in hand and travel down from the nose through the pharynx to the larynx, trachea, and bronchi—the ordinary course of progress of a common cold. In the case of the people who have acquired a relative immunity as a result of using the uric-acid-free diet, the microbe and the cold may strike them, and they may feel quite distinct evidence of some local trouble; but as the quantity of uric acid available is small, neither the microbe nor the local cold can cause any great damage, and the fire dies out for want of fuel. And this explains the observed result of diet which is the cause of my writing this paper.

Cold is thus a gout of the respiratory mucous membrane complicated by the presence of a microbe, which accounts for its power of spreading by contagion! But the microbe by itself can do nothing; it generally requires the co-operation of cold, and it *always* requires the presence of an excess of uric acid in the blood; hence it spreads among those who have available uric acid and fails to spread among those who have not.

If, then, we look upon cold as a gout of the respiratory mucous membrane, we can be certain that it will be relieved or cured by exactly the same measures that relieve and cure gout in other parts. It is disease due to the precipitation of uric acid, and it will be relieved by things which prevent this precipitation of uric acid; that is to say, by uric acid solvents; and this is just what we find to be the case. There is probably no drug which more certainly and promptly relieves an acute feverish cold than salicylate of sodium or aspirin, and it acts in this disease exactly as it does in acute rheumatism; in fact, I believe that the pathology of a common cold and of an attack of acute rheumatism are exactly the same, and the one very often passes into the other; what relieves the one relieves the other, what cures the one cures the other, and what prevents the one prevents the other.

Then, just as is the case with rheumatism, those who are taking salicylates are immune from cold, and no exposure, local or general, will harm them; and this, not because there is no uric acid in the blood (collæmia), but because it is in such good solution in combination with the solvent salicylate that it cannot be precipitated locally to cause either gout, rheumatism, or catarrh; hence both rheum and rheumatism have a common etiology and pathology; both are in part results of local cold and may be in part results of microbic action; but the uric acid is the important factor in both, and if it is removed by solvents or kept out by diet neither the cold nor the microbe can of themselves do harm.

The other important solvent of uric acid which acts in the same way is alkali, and we all know that the old treatment of rheumatism before salicylates were known was that by alkali. Here there is one physiological fact which we must bear in mind, and that is, that a low diet is equivalent to a dose of alkali, because diminishing food diminishes the acidity of the urine, and the alkalinity of the blood is at the same time increased, for diminishing food diminishes the formation of acid products in the body; hence diminished food is equivalent to a dose of alkali, and conversely it is quite useless to give small doses of alkali while feeding the patient up on full diet. If you wish small or moderate doses of alkali to have the desired effect, you must combine them with low, very low diet, even for a few hours, with complete starvation. Now I believe that no effect has ever been produced either in a common cold or in acute rheumatism on the microbes concerned, either by alkali or by low diet, and yet these measures, either singly or in combination, do undoubtedly cure both diseases. The explanation, again, is the effect of these measures on the solubility of uric acid, and the same explanation applies to the cure of both these diseases by salicylate. I think it can be shown that salicylic acid has little or no effect on microbes, and that it prevents, relieves, or cures both colds and rheumatism by eliminating uric acid, as I have always said.

With regard to treatment of a given case, you have as the result of cold and a microbe acting together a definite deposit of uric acid in a given small area of mucous membrane, say the nose and upper part of the throat. You may neglect the microbe, but you must not neglect the cold, and you must do something promptly to dissolve out the uric acid. There are two lines of treatment open to us, and these follow from the previous considerations. If there is distinct fever, we may give salicylate freely and feed up the patient on ordinary diet, or as much of the ordinary diet as he is capable of taking. If, on the other hand, there is no fever, or almost none, the best plan is to give alkali, as bicarbonate of soda, freely, and to starve the patient almost completely for one or two meals, and then put him for another 12 hours on about half his ordinary diet. At the end of that time you can slack off the alkali very decidedly, and gradually bring him back to full diet once more. It is not necessary in either case to keep on the solvent very long: you want merely to dissolve out as quickly as possible the uric acid which has been deposited in the nose and throat. A few hours will suffice to do this, and then you will prevent the further extension of the cold by stopping the solvent and clearing the blood of uric acid; because, as you remember from what has gone before, those people are immune to cold whose blood is quite clear of uric acid, that is those who live on the top of the mountain and those who are in training, but the immunity of these people lasts only as long as their blood is free from uric acid, and as soon as they go out of training or come down from the mountain all the uric acid that they have been storing in their body during their previous condition now rushes into the blood, and they fall an easy prey to local cold or the microbe or to both. Hence treatment is generally dependent on the quantity of uric acid present. Prevention follows very simply from this pathology. To be as near as possible always free from excess of uric acid means that you will be always in such a condition that neither cold nor microbe, nor the two combined, can produce a local gout of the fibrous tissues of the respiratory mucous membrane, and this explains the observed immunity of those who live an a uric-acid-free diet.

On the other hand, it may be possible to stop a cold by feeding up and tonics which clear the blood of uric acid, and thus stop the further progress of the uric acid and microbe combine.[1] The well-known dose of nitrous ether or hot wine at bedtime acts in exactly the same way—stops the collæmia. The old adage used to be "stuff a cold and starve a fever," and it is quite correct still. If there is fever, the local precipitation of uric acid is considerable, and you must use solvents, as starvation, alkali, and heat, on the one hand, or salicylates, on the other; never the two combined, for reasons which I have gone into at great length in "Uric Acid." If there is little or no fever, the local precipitation of uric acid is small, and the tonic and feeding up plan will clear the blood of uric acid (mountain climate action), and so prevent the mischief from going farther. In cases in which infection seems to be especially virulent, it may be well, in the first instance, to treat the microbe also, and this may be done simply by inhaling for a few hours a few drops of both oil of eucalyptus and salicylate of methyl from a handkerchief or inhaler, by which means we attack locally both the microbe and the uric acid. The latter drug may also be applied to the skin over the pharynx and larynx. It follows from this pathology that chronic bronchitis in the aged is a preventable disease and one from which those on a correct diet will cease to suffer and die; and my practical experiences are decidedly in favor of this conclusion, but, as with all other local uric acid troubles, the tissues lose their tendency to attack but slowly. It takes time to remove completely all the old uric acid, and until this has been done, it acts as a uric acid filter and attracts to itself any uric acid that is from time to time available in the blood; but as a practical result, just as with gout and rheumatism in other tissues, 18 months to two years on correct diet generally suffice to produce a very decided improvement.

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[1] I have suggested in "Uric Acid" (Chap. IX), that the tubercle bacillus and uric acid may form a similar evil combination, and work hand in hand through the fibrous tissues of the lungs.

PERIPHERAL CAUSES OF HEART DISEASE

A CLINICAL LECTURE DELIVERED AT THE LINCOLN HOSPITAL, NEW YORK

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Gentlemen: The causes of heart disease may be entirely outside of the heart and act mechanically through the circulation. From time to time we have had a good many cases of rheumatic disease of the heart in which the valves had become inflamed, thus bringing about a definite chain of events. There are, however, other causes of heart disease—peripheral causes, and as we happen to have several cases illustrating these, I wish to take them up for consideration this afternoon.

To understand how trouble with the blood vessels can cause heart disease we must look for a moment at the physiology of the circulation. In doing this we shall disregard the pulmonary circulation, and consider merely the systemic circulation. Leading from the heart are the arteries which divide into smaller and smaller arteries until the very small ones which we call arterioles are reached. The arterioles empty into the capillaries — very small, thin-walled vessels; and then going from the capillaries are the veins which lead back again to the heart. As I shall explain in our study of the liver, there is another system called the portal system, by which the blood from all the veins of the abdomen is carried to the liver. It is necessary to understand about these in connection with the very important subject of arterial tension.

Arterial tension is caused by the heart acting upon the blood in the arteries against the resistance caused by the arterioles. The heart by its contractions pumps the blood onward, but the small arterioles resist and in that way we get tension. If the blood was perfectly free to flow through the arterioles we would not have any degree of tension in the blood vessels because the blood would just flow off, but with the pumping of the heart and the resistance of the arterioles we get the two forces acting against each other and tension is the result. For example, suppose instead of letting the water escape freely from a Davidson syringe, you put on a very fine nozzle and kept squeezing the bulb, you will notice that very soon there will be a great deal of tension inside the syringe. If, however, you put on a larger nozzle you may work just as hard, but you will not get any tension because the fluid would flow off so rapidly.

The arteries dividing as they do into branches really have a larger capacity as they go from the heart, so that there is practically no resistance in the arteries until they get to the small arterioles.

The heart is a hollow muscle, but there is probably just as much muscular tissue in the blood vessels as there is in the heart, and the blood vessels keep continually contracting on the blood while the heart is pumping it into them. You may wonder how, inasmuch as there is a certain amount of blood in the arteries, the heart can get up tension with that amount. In answer I would say that it cannot—but that to get up tension it must have more blood. If you have only two ounces of water to use and your Davidson syringe holds two ounces you cannot get up any tension with that amount of water, and it is the same way with the heart and blood vessels. The heart cannot get up any tension in the blood vessels without using more blood than is ordinarily in the arteries. The contraction of the arterioles does not diminish or increase the capacity of the arteries to any extent. Now, where does the heart get the extra amount of blood? It gets it from the abdominal blood vessels which form a great storehouse for blood, and whenever anything happens that the heart needs more blood or any part of the body requires a greater quantity, the heart draws on the abdominal vessels for this supply.

In health the blood tension remains fairly uniform, although exercise, mental excitement, and the digestion of food cause a slight temporary increase in the arterial tension.

There are two special ways in which arterial tension causes trouble with the heart—one is acute and the other chronic.

The acute cases are those in which a sudden exertion or violent exercise causes a sudden increase in the heart's action and a sudden increase in arterial tension. Under this strain the heart becomes dilated. The cases of a chronic nature are those in which there is disease of the arteries. This is seen in a large number of cases in which either through syphilis, Bright's disease, or as the result of some infectious condition, the small arteries become contracted and lose their power of dilatation. In these cases the onset of tension is gradual, and the heart instead of becoming dilated under the strain becomes hypertrophied; the wall of the heart thickens and it works harder to overcome this resistance, and as long as it is able to do this there is no discernible heart disease except the objective sign of increase in size. By and by, however, the heart gives out and is not any longer able to do its work; the heart muscle then becomes degenerated, the hypertrophied heart becomes dilated.

We will now examine several cases of heart disease due to peripheral causes.

Case I.—This man is 30 years old and a laborer. His father died of heart disease; his mother is living and in good health. He has never had rheumatism and his health has been good except that he had malaria two years ago. He was perfectly well until a month ago. His work required the climbing of telegraph poles, and one day when he started to climb he lost his breath, gave out, and since then has been sick. When he is quiet he feels perfectly well and does not look particularly sick, but when we examine his heart we find that the apex beat is a good deal displaced to the left. On admission there were no murmurs, but the beating of the heart was infantile in character, having the same sound that is heard in the heart of a newborn child, the first and second sounds being alike. As we looked at his chest we saw what is still evident to a degree—a wave-like motion going from the base of the heart downward and outward toward the apex. The man had no other symptoms.

What happened in this case is that under a sudden call for extreme muscular effort a great tension in the blood vessels was caused which brought about a dilatation of the heart. He has no trouble with the kidneys, no discernible disease of the blood vessels, and no inflammatory disease of the heart. He has a simple dilatation due to a sudden and extreme tension in the blood vessels on the occasion of very marked muscular exertion.

He was in another hospital for three weeks, so we do not know what his treatment was, but when he came here his heart showed evidence of the administration of digitalis. This we continued for a time, until nine days ago, when we stopped all drugs and put him on the Nauheim treatment with artificial Nauheim salts. This is an imitation of a treatment for heart disease that is carried out in Germany and which is very successful in treating cases of dilatation of the heart and cases in which the heart is irregular. When this man came to the hospital the heart was irregular in force and rhythm. As I examine him at present I find the apex beat at least two inches nearer the middle line than it formerly was, the action of the heart regular, and the sounds approaching the normal in quality. He still has no murmurs.

These cases of acute dilatation of the heart are capable of complete recovery with sufficient rest and proper treatment. He may live to a good old age and never get another attack, but this should be a warning to him to seek some occupation that will not subject him to so great a strain.

Case II.—This man, G. B., is 38 years old, and a stage carpenter by occupation. His father died of old age, his mother of heart disease; he has two sisters alive and well, but one brother died suddenly. He has been a pretty heavy drinker, had syphilis about 12 years ago, but has never had rheumatism. When a child he had measles, mumps, and whooping-cough; he had pneumonia at 12, typhoid at 16, and three years ago he was in a St. Louis hospital and thinks he was treated for anemia. His present illness began about four weeks ago with shortness of breath on exertion, palpitation of the heart, and severe pains in the region of the heart.

This is not nearly as simple a case as the first one. When I saw him first I believed that his was a case of dilatation of the heart due to severe physical strain. Whether this condition was grafted on another trouble is a question for us to discuss, but there is no doubt that when he came into the hospital six days ago he was suffering from severe cardiac dilatation. The apex beat could not be felt but was believed to be well toward the axillary line. There was a marked contraction to be seen, and a well defined gallop rhythm. Gallop rhythm is very well named because it resembles exactly the sound that is heard when a horse gallops down a hard road or across a plank bridge. It is easily recognized and is characteristic of dilatation. Six days ago he was suffering from dropsy of his whole body, and all the signs that go with extreme failure of the heart. The liver was congested with venous blood until it reached to within a couple of finger-breadths of the umbilicus. The liver being congested in this way caused a condition which is named by Strumpel " pseudo-hypertrophic cirrhosis" that is a false cirrhosis, because it gives many of the symptoms of cirrhosis of the liver but is simply due to congestion of the liver with blood, for, as the heart cannot pump the blood out of this abdominal reservoir it accumulates there, and the tension being great the fluid part of the blood escapes, filling the abdomen with fluid and thus causing ascites. At present his liver is still somewhat enlarged. There was also a suspicion of a small amount of fluid in his chest. His face was so much swollen that when I again saw him the day before yesterday I did not recognize him as the same man—he has now recovered his natural features. He is, as you notice from the whites of his eyes, a little jaundiced.

All the signs in this case are those of cardiac dilatation—extreme enlargement of the heart with gallop rhythm, venous stasis, dropsy, and shortness of breath.

His occupation is such, he tells me, that he is called upon to do very hard work in a very short time, and this we can appreciate knowing he has charge of the scenery in a theater and knowing the impatience of theater-goers for the next act. We may presume therefore that this man in the hurry of moving scenery gradually acquired dilatation of the heart.

We put him to bed and gave him a half ounce of infusion of digitalis every four hours. The effect was almost magical. He had been passing a very small amount of urine (12 ounces daily) because the kidneys were so much congested that they could not work. In the next 24 hours he passed 32 ounces, in the next 24, 201 and the next, 140 ounces. At this point it seemed to me that we were doing altogether too well with our digitalis and it was stopped. During the next 24 hours its action still continued, as is likely, and he passed 101 ounces, but yesterday the amount fell to 71 ounces. In connection with this marked diuresis which was undoubtedly due simply to the clearing up of the congestion of the kidneys, he has lost practically all of his very extensive dropsy. His heart has recovered so that now the apex beat is almost in the nipple line, the sounds are normal in rhythm, and he has none of the symptoms of cardiac failure as he lies here quietly in bed.

With this man's history of a good many different diseases at different times and with the history of syphilis, it is not probable that it is a case of pure dilatation like the first case; nevertheless, the clinical picture as we have seen it during the past few days is that of cardiac dilatation. He has a soft systolic murmur at the apex and a blowing systolic murmur at the base of the heart. These were not much worse during the dilatation and still continue so they are probably connected with his anemia or with some slight damage to the valves from some previous disease.

Case III.—The two foregoing cases illustrated acute cardiac disease due to peripheral causes, but this woman illustrates a chronic cardiac condition due to chronic causes. The history is as follows: R. G., 40 years old. Her father died at the age of 90 years, and her mother at the age of 55 years, of dropsy. Her health has generally been good except for the diseases of childhood. Last April she began to be troubled with shortness of breath and suffered from pain about her heart; she also had a certain amount of swelling of the feet and abdomen. Antedating the trouble with her heart she had suffered a gradual development of disease of the arteries; the small arterioles had become hardened so that a high arterial tension developed. The heart, however, had become hypertrophied and everything went well until the heart was no longer able to overcome the resistance in the arterioles and began to give out. She then developed the signs of cardiac failure. She has no murmurs such as would be caused by inflammatory disease of the valves, but she has a sound that is caused in connection with high tension the probable explanation of which is as follows: the aortic valve which keeps the blood from going into the left ventricle closes in diastole. Now when there is a great deal of tension in the blood vessels and the heart is feeble, when it contracts to drive the blood out the tension in the arteries becomes very great, and the blood reacts and closes the valves with a great deal louder sound than normal. The accentuation of the second sound at the base is probably produced in that way—by the sudden closure of these valves which are also a little hardened in connection with the hardening of the blood vessels.

Her symptoms at the present time are the same as those we saw in the last case we examined except that they have come on very gradually and for that reason are not so marked. She has accumulation of fluid in both chests and has been aspirated—with a great deal of relief. Her liver is moderately congested and her breathing is short so that she is more comfortable when sitting up.

These chronic cases are founded upon a resistance in the blood vessels that is due to a permanent change, a thickening of the arteries which is generally but not always an accompaniment of disease of the kidneys. It is a permanent change and while we can take care of it and relieve it by drugs, still it is not susceptible of absolute cure. This case is a typical one in that the heart, blood vessels, and kidneys are all involved.

The treatment of this case must be symptomatic. We must try to relieve the obstruction in the arteries by causing a relaxation of the muscular fibers of the blood vessels by the use of nitroglycerin. We must also try to stimulate the heart to do more work by the use of digitalis and then we must give her what relief we can by mechanical withdrawal of fluid accumulating in the chest, by aspirations as often as necessary. We must keep her on a milk diet because that most favors the action of the heart and the action of the kidneys. The use of diuretics in such a case is of doubtful advisability. Her kidneys are already crippled, and it is doubtful whether after we have improved the circulation with digitalis and nitroglycerin we are justified in using those drugs that increase the excretion of urine by irritation of the kidneys. Personally I am very much opposed to it, and I believe in the long run if we take care of the circulation of the kidneys, we do more harm than good by the use of direct irritants. We must direct our efforts to the circulation and the diet.

ENDOCARDITIS IN CHILDHOOD CONSIDERED AS A SYMPTOM OF INFECTIVE DISEASES

BEING LECTURE I OF A SERIES OF CLINICAL LECTURES ON RHEUMATIC FEVER DELIVERED AT THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET, LONDON

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Ladies and Gentlemen: The subject of this demonstration is perhaps commonplace and the study hardly scientific, when we bear in mind that endocarditis is not in itself a special disease, but a manifestation of several different affections. But I do not intend to approach it in quite the routine way; I wish rather to illustrate this great truth: that endocarditis in childhood is one of the manifestations of infective diseases. By means of some clinical cases I hope to show that this truth is a living one, practical in its application to diseases of the heart, and far-reaching in its power to elucidate their problems. With its realization, the juggling with cardiac murmurs and the hair-splitting details which so often surround them with mystery, assume their true proportions, and in the broader study of the natural history of the diseases which may cause endocarditis, lose their undue prominence.

How various are the causes of endocarditis is clear when we bear in mind that the infections of rheumatic fever and of scarlet fever, the gonococcus, the pneumococcus, the streptococcus pyogenes, and the staphylococci, are all well recognized as causative agents; indeed, there are few infective diseases which at one time or another have not been recorded as causes of endocarditis; though whether as a result of their direct agency or of some secondary processes has not, as yet, been thoroughly demonstrated.

But, you may ask, Cannot endocarditis arise in childhood without an infective agent? May not some poison, produced perhaps within the body itself by a perverted metabolism, give rise to this condition without the aid of microbic infection? It would be wrong to answer this dogmatically in the negative, for the possibilities of disease appear infinite, and to-day we are the slaves of the master microbe, but this answer can I think be given. No one has shown a proof that perverted metabolism may produce endocarditis, while many have demonstrated the infective nature of the process.

I must not overlook the fact that about fifty years ago the late Sir B. W. Richardson claimed that he had produced endocarditis by injecting a 10 per cent. solution of lactic acid into the peritoneal cavity of the dog, and that he was supported in this by Rauch. Yet, later doubt was thrown upon the truth of the observation by Rayher and Johannes Müller, and, to my knowledge, the observation has never since been confirmed, and if it were, can hardly be compared to any condition that is found in disease.

It would be inopportune to enter further upon these abstruse questions, but it is well to recognize how important it is for us to ascertain whether any poisons, not due to an infective agent, can produce endocarditis. Upon the poisons of gout I would not venture to express an opinion, for in dealing with children, gout in its classical types, can be practically put aside, and it is not, I believe, common in any form. If we allow that endocarditis in childhood is the result of an infective agent, in most if not all cases, then we can proceed to investigate the history of the illness.

First and foremost in the study of this history, rheumatic fever claims our attention; it is the great cause of endocarditis, and the frequency of the occurrence is one of the strongest claims for the specific nature of the disease.

If you turn to the post-mortem records and study fatal rheumatic fever, fatal septicemia, from an infected wound for example, and fatal pneumococcus infections, you will be convinced of this: that endocarditis is almost invariable in rheumatic fever and usually absent in the wound septicemia, and that pneumonia or a suppurative pleurisy are the usual results of the pneumococcus infections. I do not deny for a moment that any one of these diseases may cause endocarditis, but in rheumatic fever only, is it the usual result.

What then is the cause of rheumatic fever? It is not my intention to discuss and argue about this question, but I will remind you that the evidence accumulated from all parts of the medical world points to an infective agent. One infective agent, I believe, to be the cause of the disease, and that a diplococcus, belonging to the great group of streptococcal organisms.

But when that question is raised—what is the cause of rheumatic fever? The answer must be a far broader one than the mere putting forward of a microbe. Why, for example, do we see in London case after case among the children of the poor? Look through the post-mortem records of a large general hospital and count the deaths from rheumatic heart disease. Recall the number of chronic "heart cases" that occupy the beds in the wards, or haunt the out-patient departments. Then some faint idea is given of the damage that rheumatic fever is doing. It maims and often eventually kills. The loss to the nation must be great indeed from the crippling effects of chronic heart disease alone.

There is the hereditary factor as in tuberculosis, but is it overcrowding or damp, or drain-poisoning or foul dust, or over-exertion, or a combination of all these, which assists the pernicious effect of the micro-organisms?

Among much that is dubious, it appears to be established now that a "sore throat" is often an early symptom of rheumatic fever, and so not infrequently a premonition of endocarditis.

This, the first clinical case, will serve to illustrate that point.

Case I.—*Faucial Angina and Rheumatic Endocarditis.*—This case is that of a girl, aged 10 years, whose mother had suffered from rheumatism. She had been brought in the first place to the hospital in May, 1901, suffering from a sore throat, with enlarged cervical glands. In June there had been epistaxis, and later arthritis and chorea developed. The heart was enlarged and there was a mitral systolic murmur. Under treatment in the hospital she improved, and was sent to Margate. During September, October, and November, the girl remained well; there was no murmur and she became fat but remained very nervous.

In January, 1902, she failed to pay me the usual visit, and in February, when her mother brought her again, there was a distinct change for the worse. I heard this history: In January she developed a severe ulcerated sore throat—the doctor had described it as almost diphtheritic—and she has not been well since. There was now once more a definite and loud systolic mitral murmur, there was arthritis, and the child had wasted. I prescribe a formalin gargle and a course of salicylate of sodium, in addition to rest; and gradually the patient improved. The systolic murmur now remains constant and there is clear proof of hypertrophy. Doubtless the dilatation of the earlier illness has been followed by an organic lesion, the result of the second infection.

This is a striking example, gentlemen, of the relation of faucial angina to rheumatic endocarditis.

The physician has long recognized this association of a sore throat with rheumatic fever, and I think that all English clinical authorities, experienced in this disease, admit the fact. In 1900, Dr. Paine and I confirmed the truth of the belief by isolating from such a throat a diplococcus, which on intravenous inoculation into rabbits produced endocarditis and arthritis. This diplococcus was identical with one which we had previously isolated from and demonstrated in rheumatic lesions.[1]

Here are drawings that were made from the actual microscopic sections. They are faithful representations. They show the diplococcus in the human tonsil (Fig. 1), in the mitral valve, synovial tissue, rheumatic nodule, and pleural (Fig. 2) and pericardial exudations (Fig. 3). Here again are macroscopic preparations of experimental endocarditis, both simple (Fig. 4) and malignant (Fig. 5);[2] and of human rheumatic endocarditis, simple (Fig. 6) and malignant (Fig. 7). Under the microscope you will see the diplococcus in an inflamed human mitral valve.

There is but little doubt that the micro-organisms gain access to the valves by making their way from the tonsils into the blood stream and then they survive and multiply in those tissues which favor the rheumatic infection.

Three morbid changes that they produce in the cardiac valves are worth remembering: (1) swelling of the subendothelial tissues; (2) cellular proliferation; (3) necrosis of portions of the valve.

Here are drawings which illustrate these changes in the human valve (Figs. 8 and 9), from their early commencement to the formation of the vegetation; and to complete the pathological picture,

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[1] The researches of Meyer, Menzer, and Allara, in 1901–1902, have done much to elucidate this point.

[2] The drawings and photographs of the lesions not illustrated in this paper, have been already published in the Lancet, September, 1900, and May, 1901, and Path. Soc. Trans., 1901 and 1902.

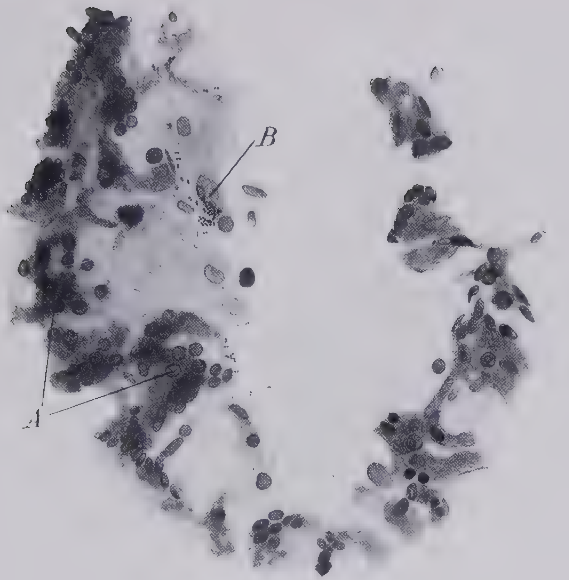


FIG. 1.—A section through a crypt of human tonsil from a case of rheumatic angina, showing diplococci. *A*, Inflammatory cells; *B*, Diplococci.

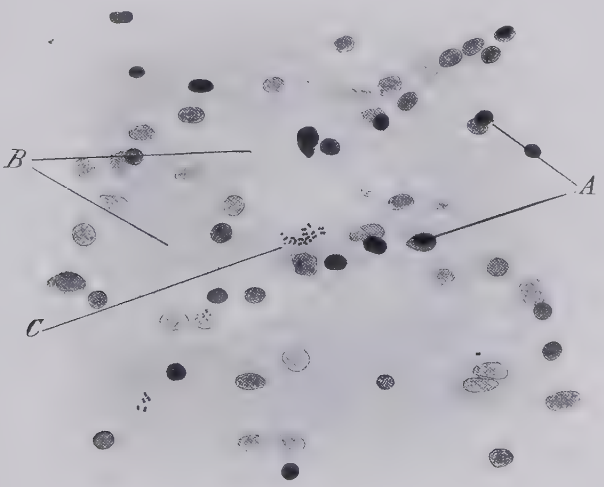


FIG. 2.—A film preparation of the exudation from a case of rheumatic pleurisy. *A*, Leukocytes; *B*, Fibrinous exudation; *C*, Diplococci.

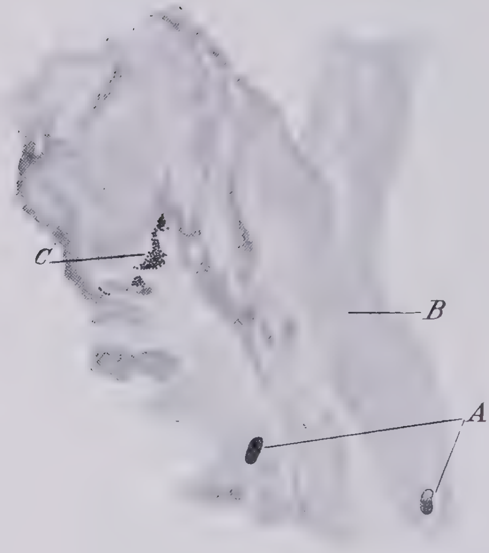


FIG. 3.—A film preparation of the pericardial exudation from a case of rheumatic pericarditis (human). *A*, Leukocytes; *B*, Fibrinous exudation; *C*, Diplococci.

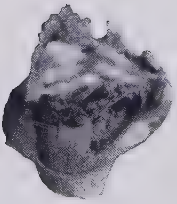


FIG. 4.—Simple endocarditis of the mitral valve experimentally produced in a rabbit.

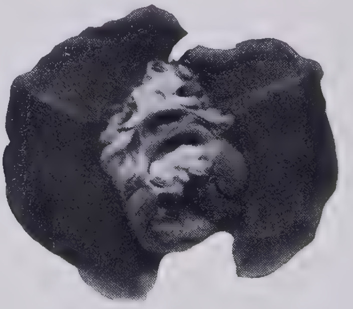


FIG. 5.—Malignant rheumatic endocarditis of the mitral valve experimentally produced in a rabbit.

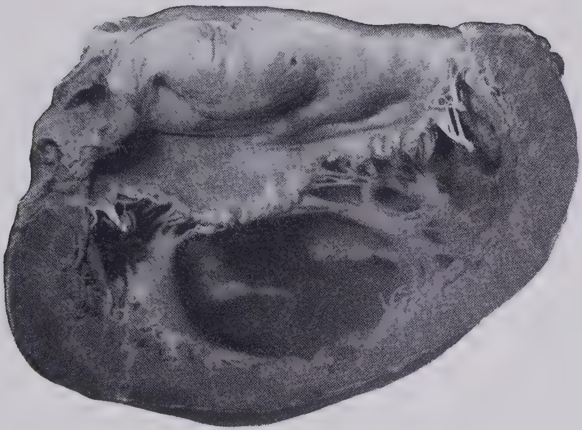


FIG. 6.—Simple rheumatic endocarditis of the mitral valve (human).

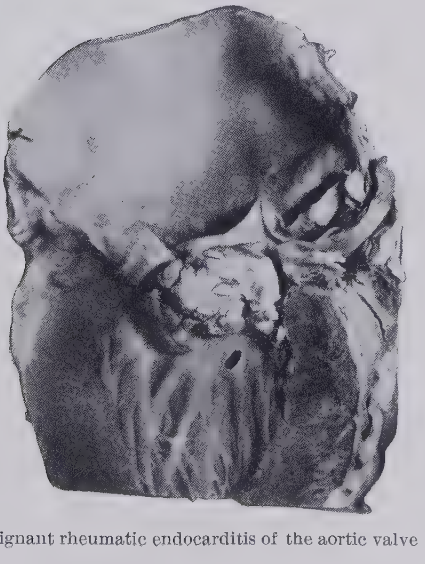


FIG. 7.—Malignant rheumatic endocarditis of the aortic valve (human).

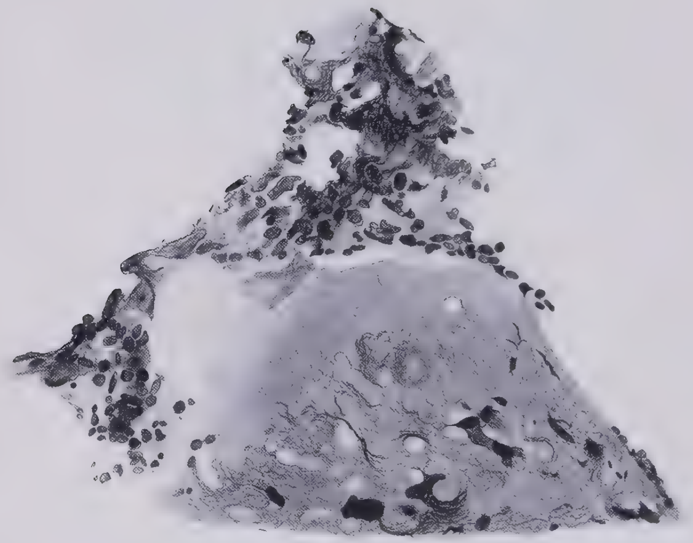


FIG. 8.—Early endocarditis of the mitral valve ; a section through a vegetation. There is in the upper part of the figure free cellular exudation, and among the cells under higher magnification diplococci well shown (human).

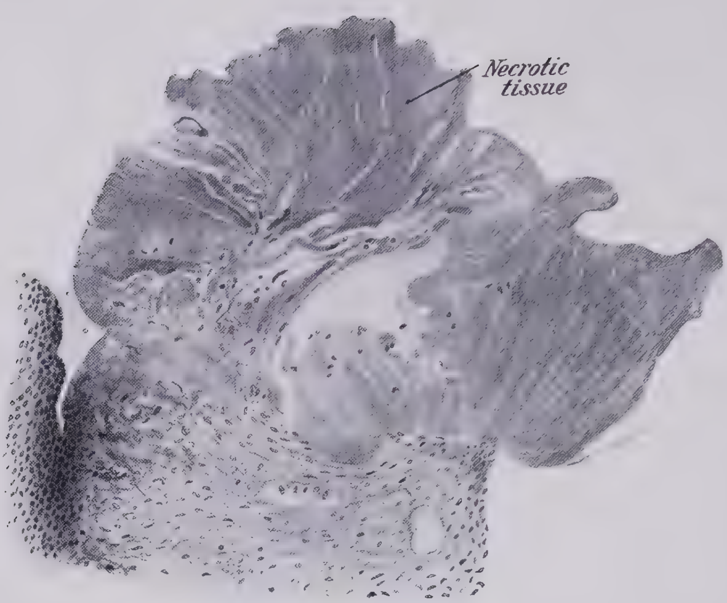


FIG. 9.—A section through a vegetation in simple rheumatic endocarditis. The stage is more advanced than that in Fig. 8. There is much necrotic tissue, but no diplococci, and at the base reparative changes are commencing (human).

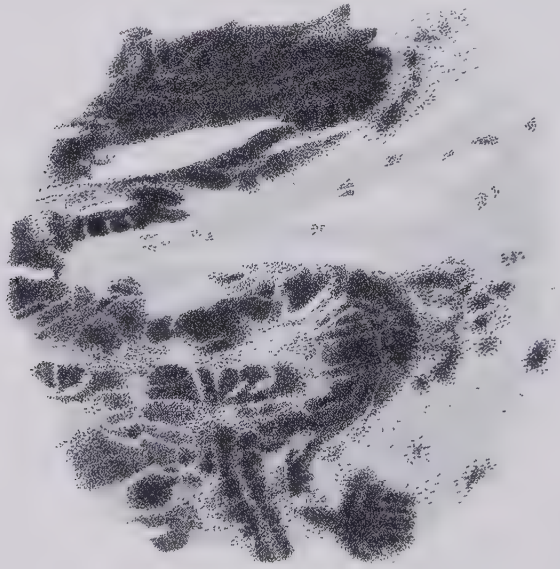


FIG. 10.—A section through a vegetation in malignant rheumatic endocarditis. The necrotic tissue, in striking contrast to Fig. 9, contains myriads of diplococci (human).

here again is the macroscopic specimen of rheumatic endocarditis (Fig. 6).

There are two points to which I would direct your especial attention. The first is, that a cap of fibrin is not an essential part of a vegetation; and the second, one of great importance, that the diplococci are, as a rule, rapidly destroyed at the site of the lesion. When in man death occurs, not we know as a rule from acute endocarditis, but from concomitant pericarditis, few, if any, diplococci, will be found in the necrotic part of the vegetation upon the valve.

Contrast that drawing of the simple vegetation (Fig. 9) with this one from a case of malignant endocarditis (Fig. 10), and note the myriads of micrococci in the latter. Contrast also the macroscopic specimens, illustrating the two types of endocarditis, both in man and the animal. (Figs. 4, 5, 6, and 7.)

In childhood more than one valve is often affected. The damage, as Sir Thomas Barlow has observed, may not be great, but more than one valve shares in the process. The order of frequency is first the mitral then the aortic, next the tricuspid and very rarely the pulmonary valve. When two valves are damaged the lesions most frequently combined are those of mitral and aortic regurgitation.

Why is the mitral valve the most frequently damaged? I believe it to be because it is the most complex in structure and contains minute blood vessels, in which the diplococci travel to the tissues of the valve. The aortic comes next I believe, in part, because of its proximity to the mitral; this enables the micro-organisms to spread from the one to the other. I do not believe the difference of the blood in the right and left chambers of the heart has anything to do with the preponderating frequency of the endocarditis upon the left side, because I think that the diplococci gain access to the valves, not from the blood in the chambers of the heart, but from that in the capillaries of the coronary arteries which always contain arterial blood. For the same reason I traverse the sweeping assertion which is sometimes made, that only the left side of the heart is affected by rheumatic endocarditis. Here, indeed, is proof to the contrary. In 1898, I made an analysis of 150 fatal cases of rheumatic fever, and in 149 of these the mitral valve was damaged, in 51 of these the aortic valve was damaged, in 36 of these the tricuspid was damaged. These figures were obtained from necropsies made by many different pathologists and they are quite free from any bias. It is clear too, that if the infection takes place through the coronary circulation, the right side will be sure to suffer in a certain percentage of cases.

Finally, before we turn to the clinical side of the subject again, it should always be remembered that there are two types of morbid changes in rheumatic fever: those due to the local lesions, which have already been detailed, and those which result from the general toxemia, the exact nature of this toxemia being at present not known.

When the clinical side of the subject is approached, the first question will be: What are the earliest clinical evidences of rheumatic heart disease? The answer is: A little fever, perhaps some palpitation, and pain, possibly even scanty breath on exertion, and a quickened low tension pulse; and these physical signs: (1) an outward displacement and feebleness of the cardiac impulse; (2) an increase in the area of the deep cardiac dulness; (3) a diminution in the length of the first sound, and an accentuation of the pulmonary second sound at the base; and (4) in some instances the development of a soft systolic murmur internal to the left nipple.

The interpretation is this: rheumatic fever causes a dilatation of the heart.

This fact has been repeatedly emphasized, and its importance insisted upon by Dr. D. B. Lees during the last ten years, and I will show you some tracings upon glass, which I made at the time I had the honor of working with him upon this subject.

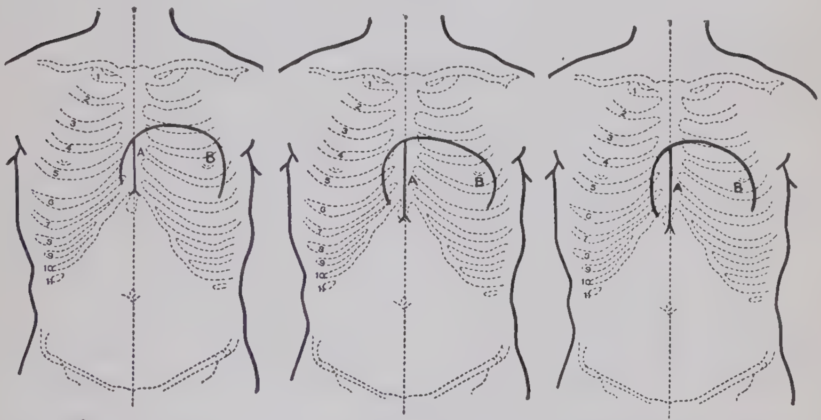
To-day, it is not possible in the time at our disposal, to put before you the myocardial lesions that occur in rheumatic fever; another demonstration will be necessary. Nor would it be advisable to divert our attention from the subject of to-day—endocarditis. But, this point we must recognize: *that the early clinical evidence of rheumatic heart disease is often myocardial rather than endocardial.*

A word now about these tracings: The limits of the deep cardiac dulness were carefully percussed and outlined upon the chest wall, then transferred to tracing paper, reduced to scale and painted upon glass, by my friend Dr. Ransome. Each observation during the attack of rheumatic fever, was made independently of the other, and each outline is painted in a different color. The phenomenon of dilatation can in this way be illustrated upon a screen.

I cannot show you clinical cases with this condition of dilatation (these glass slides, Figs. 11, 12, and 13, must suffice), for such cases require rest in bed, and cannot be well dwelt with in an out-patient department.

This early dilatation may entirely disappear—not a trace need remain; and so the next clinical question is one of much interest. It asks whether it is possible to distinguish dilatation from early endocarditis?

Fig. 11. Fig. 12. Fig. 13.



Illustrating variations in the degree of dilatation of the heart. Fig. 11.—The condition on admission—slight dilatation. Fig. 12.—After a relapse of rheumatism—increased dilatation. Fig. 13.—Convalescence—diminution of dilatation. A, The midsternal line; B, the nipple. The patient eventually recovered without a trace of permanent damage.

If by this is meant—can we draw a hard and fast line between the two conditions, I believe not; for, in the first place, a bruit may occur without endocarditis; in the second, endocarditis may occur without a bruit; and lastly, both dilatation and endocarditis are often associated. Although, we cannot be absolutely certain of distinguishing between early dilatation and early endocarditis, we can, by experience, judge pretty accurately of their occurrence. Exceptional cases, as in everything medical, may leave us in doubt; but as a rule, if we are not in too great a hurry, a correct conclusion can be drawn.

When the cardiac area is considerably increased, and yet there is no systolic murmur or only a faint one, which is heard most clearly internal to the nipple; and when the enlargement of the area of cardiac dulness diminishes and disappears without the appearance or persistence of a murmur, it is certain that there has been cardiac dilatation, and that there has not been, in all probability, endocarditis.

Pericarditis, you will remember, in rheumatism is usually detected by the friction rub. If the area of cardiac dulness is considerably increased, but the cardiac sounds are clear, and there has been no friction rub, you may rest assured that the enlargement of the cardiac area is due to dilatation and not to pericardial effusion.

When a systolic mitral murmur remains persistent and can be traced toward the axilla, and when the second sound at the impulse is persistently reduplicated, and the heart hypertrophied, then we know that whether or not there has been dilatation, there is now organic valvular disease. The reduplication of the second sound, if indeed it be a reduplication, points to a thickening of the mitral segments and is of the nature of a presystolic murmur.

In some cases it is possible to trace the development of endocarditis subsequent to an already existing dilatation. If it be of the mitral valve, then the signs of dilatation, which I have already detailed, being present, you notice one day an abrupt alteration in the character of the first sound, which becomes indefinite and hard to detect. And then later, a sharp systolic whiff traceable toward the axilla is heard. If this whiffing murmur seems far from your stethoscope at a deeper level than the cardiac sounds themselves, the mitral valve is, I think, inflamed.

On two occasions, within a space of 48 hours, I have traced the early development of aortic endocarditis. The stages are these: first of all the aortic second sound becomes faint—you have as it were to search for it and to ask for silence in the room; then you hear a soft diastolic blow and you know that the lesion of aortic regurgitation has resulted. It is astonishing in childhood how rapidly the character of the collapsing pulse will appear after the aortic valve shows signs of incompetency.

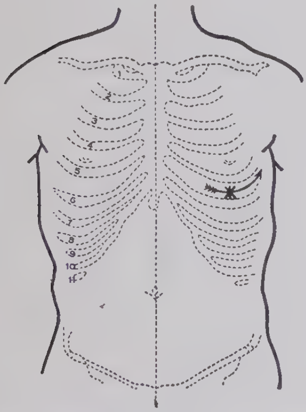
Case II.—*To Illustrate Mitral Endocarditis.*—This child, H. G., aged 11 years, has been wasting for three months—a very common history with rheumatic children. Two years ago there was an indefinite history of pains in the legs and sweating.

You will notice that the systolic mitral murmur is extraordinarily loud and following the murmur is the curious reduplicated sound which, apart from the bruit, convinces me that the lesion is organic. The hypertrophy is very definite. When listening to such a murmur it is well to recall that the loudness is no index of the severity of the lesion.

The figure shows the position of the early presystolic murmur (Fig. 14) and the direction of conduction of the mitral systolic murmur.

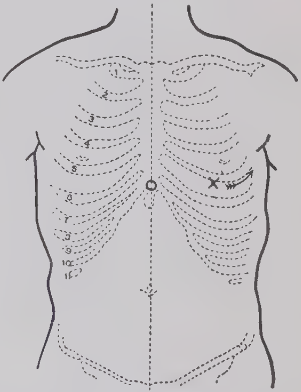
Case III.—*To Illustrate combined Mitral and Aortic Endocarditis.*—This boy, W. A., aged 10 years, was first seen in July, 1901, and had already suffered from three previous attacks of rheumatic arthritis. He came to the hospital the picture of a severe case of aortic regurgitation, pale, breathless, and nervous, with the vessels throbbing visibly at the temples, with capillary pulsation, and a pulse which, on raising the arm, collapsed to such a degree as to be almost imperceptible. The temperature was 100° F.

Fig. 14.



✱ Mitral presystolic murmur. ⟶ Mitral systolic murmur.

Fig. 15.



○ Aortic diastolic murmur. × ⟶ Mitral systolic murmur.

He remained in the hospital for seven weeks. Since then I have never lost sight of him, and I managed to guide him through last winter and all this year—aided by a most sensible mother—without another attack. At present he is better than he has been for some years, but the damage is irreparable.

Fig. 15 shows the position at which the diastolic aortic and systolic mitral murmurs are most clearly heard.

We have now gradually drifted from the acute endocardial lesions to the chronic ones, and those who have studied the drawings of early endocarditis, will easily understand how those deformities are caused which form the basis of the study of chronic valvular disease of the heart.

Let me once more show you the rheumatic vegetation in the necrotic phase (Fig. 9). You will see that the base is formed by the fibrous tissue of the valve, infiltrated with living and active cells. Step by step this necrotic area is invaded by fibroblasts, and gradually this young imperfect connective tissue contracts and distorts the segments of the valve.

This photograph[1] shows you the early change in a chorda tendinea of the mitral valve of a rabbit. The swelling upon it is a connective tissue swelling, and it was caused by the presence of the diplococcus. All that was done to make this specimen, was to remove the mitral valve with two chordæ attached—one was the damaged chorda, the other was natural—and to spread it out flat upon a glass slide. It was then photographed under a low power of magnification. You must all, I think, allow that this is a specimen of much interest, for it gives you a glimpse into the early stage of that process which leads to shortening and deformity of the chordæ tendineæ, and gives you an insight into the developmental stage of mitral stenosis which cannot be gained even in the post-mortem room.

Once again, let us interpret some clinical facts by the study of these processes of healing.

It is not very unusual to find that after some months a systolic mitral murmur may disappear, and it is quite conceivable that the repair of a simple endocarditis may be all but perfect. You do not marvel over the complete disappearance of a rheumatic nodule, and the two lesions are closely analogous.

Not only may a mitral murmur disappear, but an aortic murmur also.

But there is another reason for the disappearance of a murmur in acute rheumatism, a more usual one, which is this: there has been dilatation of the heart and the murmur has been produced by relative incompetence. When the dilatation subsides, the murmur disappears.

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[1] Reproduced in the Lancet, September, 1900.

This phenomenon of the disappearance of a murmur has a very special importance in the estimation of the value of methods which are employed for the treatment of acute endocarditis. It is a clinical fact which authorities upon the subject of heart disease have long pointed out, and it is not a rare occurrence. Yet, from time to time, it comes as a revelation to some one who is studying the treatment of heart disease. He naturally ascribes the result to the treatment and the treatment is perhaps magnified by others into "a cure." But it is "kindly nature" probably who should receive that thanks, for all treatments which include rest will meet with the same result in a certain percentage of cases, and no drug is certainly known to influence acute endocarditis. I have under my care at present two examples of this disappearance of the murmur. The one was treated with an alkaline solution of quinin, and the other with anti-streptococcus serum.

Case IV.—*To Illustrate the Disappearance of Cardiac Murmurs.*—This boy, aged 6½ years, came here first in February, 1902. There is a family history of rheumatic fever, and before this illness the patient had suffered from vague pains in the limbs. In December he developed a sort throat; then followed inflammation of the mitral valve, hemichorea, and arthritis. He was admitted to the hospital and whilst there the aortic valve was attacked.

In May he came to see me again with another attack of rheumatic fever, was readmitted to the hospital, and there suffered from a severe pericarditis.

Now, to-day, though he was so very ill in June, you will be surprised when you examine his heart. There is a good impulse. I cannot hear the aortic regurgitant murmur—which had been audible for some months—and only with difficulty can I detect any mitral bruit.

The converse to the disappearance of a murmur is also met with and may be a cause of disappointment and sometimes, without due reason, of discredit to the medical attendant. I mean that a cardiac murmur which was absent during the acute attack, may make its appearance some months later. Now the causation of cardiac bruits is not at all easy to understand, in spite of the multitude of writings about them. Consider for a moment these two specimens, from a recent investigation by Dr. Paine and myself. In this specimen, you see a large vegetation upon the mitral valve (Fig. 5), yet during life no murmur was audible over the rabbit's chest. But with this other one, during life, there were loud systolic murmurs, which led us to predict and rightly, that both the aortic and mitral valves were affected. You see exuberant vegetations upon both of them. If such gross lesions produce bruits in one case and not in another, it does not seem to me difficult to realize that a deformity in a cardiac valve, produced by the process of healing, may be the cause of the appearance of a bruit which was absent during the acute phase.

It is impossible now to make more than a few discursive remarks upon chronic heart disease. You have already seen some types in the early stages, and in conclusion, let me show you a case of mitral stenosis and a case of aortic stenosis (Cases V and VI). Mitral stenosis of an advanced type is not common in childhood, but the condition is one of the very greatest interest in the scientific study of rheumatic fever. This question will make that clear: Is stenosis the result of the healing of a single severe attack of acute endocarditis, or is it the result of a persistent smouldering infection? In the answer to this I feel convinced there lies hidden much that we need to know about rheumatism. It was Dr. Stephen Mackenzie, I believe, who pointed out that sometimes before the characteristic bruit of mitral stenosis appears, there may be a complete absence of murmurs, that is to say, a systolic murmur previously present, disappears, and then after some time has elapsed a presystolic murmur is heard.

Case V.—*To Illustrate Mitral Stenosis.*—This little girl, aged 10 years, exemplifies very distinctly the condition of pure mitral stenosis. There has never been a definite history of rheumatic fever, nothing more than occasional sore throat and some aching pains in the knees and elbows. For three years at least the heart has been damaged.

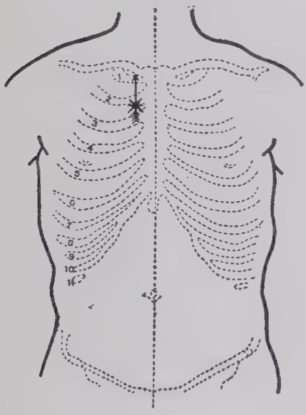
The presystolic thrill, the rumbling presystolic murmur, the disappearance of the second sound at the impulse, the loud banging pulmonary second sound, all point to an advanced mitral stenosis with little, if any, regurgitation.

It is remarkable how latent the rheumatic manifestations are apt to be in these cases of mitral stenosis, and the well-known frequency with which mitral stenosis is met with in females as compared with males, is also very curious. The child, you will perceive, is stunted, for the output of blood from the left ventricle is small.

Case VI.—*To Illustrate the Bruit of Aortic Stenosis.*—This little girl, C. E., aged 8 years, is an interesting case. There is a doubtful family history of rheumatism, but no personal history. The only illnesses the child has suffered from have been mild attacks of scarlet fever and whooping-cough. All the evidence that I have been able to obtain points against a congenital lesion of the heart. I show her to-day as illustrating the bruit of aortic stenosis.

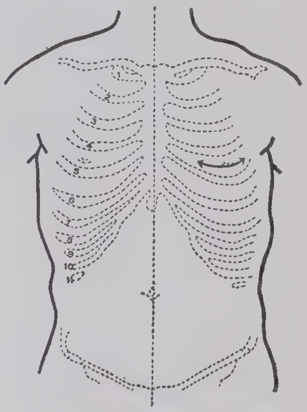
In the second intercostal space on the right side close to the sternum (Fig. 16), you hear a loud systolic murmur, harsh in character, traced very definitely into the large vessels at the base of the neck, and becoming less audible when traced toward the impulse. There is a systolic thrill over the upper part of the chest. The child is stunted and fragile and suffers from attacks of dyspnea and cardiac pain.

Fig. 16.



↑✱ Aortic systolic murmur,

Fig. 17.



⟷ To-and-fro mitral murmur.

An error that is sometimes made by those who are not well acquainted with endocarditis in childhood, is to mistake a to-and-fro murmur heard over the impulse for an indication of disease of the aortic valve. Such a murmur in the adult is as a rule aortic in significance, but in the child, it points to mitral damage.

Case VII.—*To Illustrate the To-and-Fro Mitral Murmur.*—This boy, A. B., aged 11 years, has suffered from rheumatic fever off and on since he was five. When I first saw him there was dilatation and hypertrophy of the heart, a subcutaneous nodule on the right olecranon, and a stiff neck. The to-and-fro mitral murmur is very plainly heard immediately around the impulse. It indicates a mitral lesion, the aortic valve being intact (Fig. 17).

Edema is not so common in the child as in the adult, but edema of the face is, I fancy, more common. Partly, I believe, because rheumatism in its severe visceral types damages the kidney, and partly because there is a liability to venous thrombosis of the large cervical veins.

It is remarkable how little distress even grave heart disease may cause a child, but the great test of healthy childhood, the power to rush about, in season and out of season, usually tells a pathetic tale. Children afflicted with morbus cordis are easily terrified, and often highly nervous; how much this is due to active rheumatism, and how much to impaired cerebral circulation, I do not know.

Last of all, but perhaps the most important of all, there is the question to be answered: Why, since compensation is so good in childhood and the valvular damage often slight, does the heart break down so often? The answer is that fresh rheumatic attack is the cause of the breakdown. I do not mean that overstrain is never responsible, but in an analysis of 100 fatal cases of rheumatic heart disease I found undoubted evidence of a fresh attack of rheumatic fever in 86! I doubt, in spite of the teachings of authorities upon heart disease, whether the true significance of those numbers has been thoroughly recognized. Their immense importance in considering the prognosis of rheumatic heart disease cannot be overlooked. If those numbers could speak, they would say: "Until you know more about the determining causes and the life history of rheumatism, and more about the infective agent in rheumatism, you cannot forecast rheumatic heart disease in childhood." Who can say that a case of slight mitral endocarditis of to-day may not appear next spring as a hopeless case of general pericarditis?

This concludes the demonstration, and if I have succeeded in leading any one present to-day to a clearer conception of the infective nature of endocarditis in childhood, I shall feel that the time has been well spent. My sincere thanks are due to Dr. F. G. Penrose and Dr. A. E. Garrod, for allowing me to make mention of those cases which while in the hospital were under their care.

THE ETIOLOGY AND DIAGNOSIS OF VALVULAR AFFECTIONS OF THE HEART

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INASMUCH as in the past a close comparison between clinical signs and post-mortem findings has laid the foundation for whatever success has been reached, thus far, in the diagnosis of valvular diseases, so also in the future advances will be made along the same lines. It is surprising, however, to notice that, with all the wealth of literature on this subject before us, few individuals, comparatively, have contributed positive criteria, and that our knowledge has been gleaned from a narrow field. It is for this reason that our progress has been so slow.

To the Paris hospitals, as in many other matters of cardiac pathology, we owe the first debt of gratitude. After them we are indebted mainly to Guy's and to St. Bartholomew's Hospitals, of London, and later to the Charité, of Berlin. Quite recently the Massachusetts General and the Johns Hopkins Hospitals have contributed more than other institutions or individuals in this country to what we actually know to-day. And in the future we shall depend almost entirely on the larger hospitals for further elucidation of the problems in valvular affections that are now confronting us; for it is only in these institutions that we can collect the necessary data in sufficient quantity for profitable use, and at the same time secure the intelligent co-operation of clinicians and pathologists in utilizing the data to determine the precise value of clinical signs.

In the present paper I shall make use of the material that has come to me in my New York hospital experiences, and, though my contribution is modest, the number of autopsies being much smaller than some that have been utilized by my confrères, it is, I believe, the first contribution of the kind from New York City. It has the value of a study of unselected cases—cases as I have actually found them under, I believe, as advantageous opportunities for study as our city has afforded.

Within the limits prescribed for this paper I shall leave unsaid many things that might properly be said on these topics, restricting myself to certain facts, figures, and critical remarks, which I hope will help to remove some of the haze that at present surrounds the symptomatology of endocardiopathies, so that their diagnosis will be facilitated. I shall take up these affections in order, beginning with those of the mitral valve.

Mitral disease is exceedingly common. Sperling,[1] in 100 cases of combined valvular lesions, found that the mitral was affected in 99 (99 per cent.). In 44 cases of combined valvular lesions I found the mitral affected in 89 per cent., most often with aortic disease, less often with tricuspid disease, and very rarely with the pulmonary valve disease. As a single-valve lesion, Sperling found it in 78.5 per cent. in 200 cases; I in 33.3 per cent. in 27 cases; but the small number of my cases as compared with Sperling's does not justify me in basing any particular value on this latter percentage.

Lithemia, of course, is the most potent cause of the organic forms of mitral endocardiopathies, but the causes of the inorganic or relative varieties are manifold.

Many of the young men who train for athletic contests develop a mitral insufficiency during the strain of the course. In fact, I presume that it is a common affair after a boat-race or a running match, and also after an attack of infective disease. But, inasmuch as these incidents or accidents, whichever we call them, do not cause death, we seldom see them at autopsies. However, I have found them at the post-mortem table in fatty-heart patients, and in aneurism of the arch of the aorta, when the valve orifice had been dilated, the leaflets failing to adjust themselves to the enlarged opening.

But, after all, this relative insufficiency is often (not always, of course) a temporary condition that will mend with rest from the strain of athletics, and perhaps after symptomatic treatment; while the forms that result from infections, like diphtheria or the continued fevers, influenza, etc., will improve synchronously with improvement of the general system. And yet in aneurism, when pressure causes insufficiency, or in adherent pericardium, so long as the adhesions persist, the heart will be dilated and the insufficiency will become chronic. In my opinion, however, organic insufficiency due to lithemia or allied conditions tends to stenosis, because all infiltrations of inflammatory matters contract if they do not disintegrate.

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[1] Gibson's Diseases of the Heart, London, 1898.

According to my tables, the diagnosis of mitral insufficiency is made in about three-fourths of the cases. It is the one affection that is most certainly diagnosticated, for the diagnosis is comparatively easy—the three cardinal signs being: (1) A systolic murmur at the apex conveyed to the left; (2) accentuation of the second pulmonic sound; and (3) increased transverse dulness, indicating dilatation or hypertrophy of the left ventricle, or both. There is also weakness of the second aortic sound, and in advanced cases hypertrophy and dilatation of the right ventricle.

If there is also mitral stenosis, we may expect, in a certain number of cases, a systolic thrill somewhere about the apex in from 15 per cent. to 60 per cent., and a presystolic murmur in from 10 per cent. to 30 per cent., or perhaps more.

In failing compensation the first two signs will fail, and, as the others are not pathognomonic, the diagnosis is then problematical.

Obstruction at the mitral, or left auricular ventricular, orifice is often called stenosis, although obstruction is the better term, because it is more comprehensive; for there can be, and often is, obstruction without any narrowing (stenosis). On the other hand, there can be no stenosis without obstruction. In fact, obstruction with dilatation is not very uncommon, and it may cause a loud, obstructive murmur.

This statement that I make in relation to the distinction between obstruction and stenosis applies to all the valves.

In the order of frequency, Walsh puts mitral obstruction fourth on his list. I have not only found it fourth in my pathologic series, but also in a series of 50 clinical cases taken from my clinical registers at the Post-Graduate Hospital. But as mitral insufficiency, in my opinion, precedes mitral stenosis, the latter should be less frequent. But they coexist when stenosis has developed, and they coexist according to my pathologic records in 70 per cent. of mitral diseases. On the other hand, advanced stenosis, when the opening is reduced to a cleft or slit, is uncommon, for in 71 cases of mitral stenosis that I have collected from the literature, including my own, this degree of stenosis occurred in only about one-half of them.

But the diagnosis of mitral obstruction is beset with many difficulties, as our pathologic records show. In 48 cases of obstruction, as proved by post-mortems in the Massachusetts General Hospital, only 23, or 48 per cent., were recognized during life.[1] In my 27 cases, on the other hand, this lesion was not noted at all in 19, or 70 per cent.; the signs were either not heard or were misinterpreted in 4, or 15 per cent.; they were correctly heard and interpreted in 4, or 15 per cent.

In this connection we may bear in mind that up to the year 1871, according to Fagge,[2] only 28 instances had been reported in which a presystolic murmur noted during life had been verified by post-mortem. And, again, Fauvel[3] only discovered this murmur in 1843, and doubtless its importance has been underestimated.

But in Fagge's own series of 47 cases the presystolic murmur had only been noted in 7, or 15 per cent.—a percentage, it will be noted, that accords precisely with my own. However, the fact must not be overlooked that Samways,[4] in 156 cases of mitral stenosis proved by autopsies at St. Bartholomew's Hospital, found that the presystolic murmur had been audible in about 60 per cent., and that Hayden,[5] in 15 autopsies, found the presystolic murmur in 12, or 80 per cent. It is difficult to reconcile such conflicting evidence. However, it must be remembered that there is a personal equation even in the valuation of auscultatory phenomena.

In 20 cases I found the following:

A simple mitral presystolic	murmur	in	3, or	15 per cent.
“ “ “ systolic	“	“	4, or	20 per cent.
“ systolic and presystolic	“	“	5, or	25 per cent.
No murmurs		“	8, or	40 per cent.
			20, or	100 per cent.

So that from my figures the grand total of murmurs in the diastolic interval was 40 per cent. Fagge found 28 per cent. of diastolic murmurs apart from the presystolic, making his total of murmurs in the diastolic interval 43 per cent.

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[1] Cabot, Physical Diagnosis, New York, 1900, p. 163.

[2] Fagge, Guy's Hospital Reports, March 16, 1871, 31st ser.

[3] Fauvel, Arch. Gén., 1843, fourth ser., tome i, p. 5.

[4] Samways, Brit. Med. Jr., 1898, i, p. 36.

[5] Diseases of the Heart, Dublin, 1866.

From my own experiences, proved, as they have been, by post-mortem evidences, in conjunction with those of other observers similarly proved, I find that the following points bear on the diagnosis of mitral obstruction:

(1) In certainly 15 per cent. there is a simple presystolic murmur. In fully 25 per cent. additional, and perhaps even more frequently, there is heard some other murmur besides the presystolic in the diastolic interval. Thayer,[1] of Baltimore, found the presystolic murmur in 81 per cent. of his cases.

(2) The maximum intensity of the bruit lies above the apex, usually over the fourth left interspace or at the fourth sternocostal articulation, but it is not absolutely fixed. In Dr. Tyson's case of presumed mitral stenosis (the patient being still alive) it was heard at one time in the fourth left interspace below the nipple, subsequently in the fifth left interspace below the nipple, and later still in the upper part of the fifth interspace inside the nipple.[2]

(3) The bruit is of short duration, and, as a rule, is not transmitted to a distance, and it runs up abruptly into the first sound, often (in 68 per cent., Thayer) with a pronounced shock.

(4) The second sound at the base at the pulmonary area is intensified. Occasionally with the second sound at the base there is a murmur heard over the left auricular appendix.

(5) In my cases a thrill was present in from 10 to 35 per cent. Thayer found a thrill in 54 per cent.

(6) Another sign is contemporaneous hypertrophy of the left auricle and of the right heart.

(7) There should be a history of a preceding mitral insufficiency of organic character.

Aortic disease is less common than mitral, for if we accept Sperling's figures in 200 cases in which only one valve was affected it was the aortic in only 40 cases, or 20 per cent. And in his 100 cases of combined lesions it was 89, or 89 per cent., as against 99 per cent. for mitral affections. In my 44 cases of combined lesions the aortic was involved in 39, or 89 per cent.; so that there is a very close association between these two valvular affections.

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[1] Medical Record, January 18, 1902.

[2] Phila. Med. Jour., May 18, 1901.

Of the two varieties, insufficiency and obstruction, the former is the more common.

Aortic insufficiency develops so insidiously that it is apt to exist a considerable time before it is recognized. Indeed, for a while there may be no physical signs, subjective symptoms being the first indications to warn the patient.

In established aortic insufficiency there is the "long heart," in which the apex extends as far as the sixth, seventh, or eighth interspace, occasionally reaching the axillary line. There may be a thrill, or a thrill may be absent. Besides the characteristic Corrigan or water-hammer pulse, there may be the capillary pulse, or perhaps in some instances Quincke's venous pulse, in which the blood is forced through the capillaries into the veins.

The diastolic murmur is heard clearly in the second left interspace, sometimes in the second right interspace close to the sternum, or between these points; but being propagated downward toward the ensiform appendix or the apex of the heart, it may be heard with greatest intensity in the middle sternal region or at the base of the ensiform appendix, usually to the left of the sternum (however, sometimes to the right of it), in the line of the fourth left costal cartilage. In the last named instance the murmur is perhaps conveyed by the solid mass of cardiac tissue that intervenes between the aortic valve and the listening ear, for solid tissue, bone, and the flowing current of blood may each conduct sound successfully.

In uncomplicated cases of aortic insufficiency it has been found by W. S. Thayer,[1] from a study of 74 cases at the Johns Hopkins Hospital, that a presystolic or a mid-diastolic murmur limited to the region of the apex occurs in about one-half of the cases. This murmur, discovered by Flint in 1862, differs in no way, he thinks, from that commonly observed in the true mitral stenosis, except that it is usually of moderate intensity. It is, however, rarely associated with a "tapping" systolic impulse and a "tapping" first sound, which are characteristic of mitral obstruction. In the absence of these signs and with a large pulse, the presystolic murmur at the apex should lead to the suspicion of aortic insufficiency. In about one-half of these cases of the Flint murmur there is arteriosclerosis.[2] The close association of aortic with mitral disease led to the theory that the Flint murmur in aortic insufficiency is due to the current of blood impinging on the inferior leaflet of the mitral valves; but although this theory may serve as an explanation in some cases, it is evidently not of universal application, for, according to Thayer, in 17 out of 33 cases the mitral was healthy, and Cabot has, to a certain extent, verified this statement by his personal experiences at the Massachusetts General Hospital. In dilatation of the ascending arch, which is common in organic aortic insufficiency, the murmur may be carried far to the right, although the dilated arch does not necessarily occasion dulness on percussion.

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[1] Amer. Jour. of the Medical Sciences, November 19, 1901.

[2] Medical Record, May 11, 1901.

Aortic insufficiency is recognized with a fair degree of accuracy. In my 37 cases it was noted in 23, or 62 per cent. In 65 cases proved by autopsies at the Massachusetts General Hospital it was recognized during life in 44, or 67 per cent. (Cabot). There is, however, great difficulty in distinguishing between true aortic insufficiency and aortic aneurism, particularly as in aortic disease there is often dilatation of the arch (the false or fusiform aneurism).

The points of difference are that the bruit and thrill of the latter diminish as the ear recedes from the so-called aortic area, while in true aneurism the bruit and thrill increase. Also there is more or less dulness over the aneurismal sac, certainly if it contains any fibrin. In aneurism, too, there is no Corrigan pulse.

Aortic obstruction is a tolerably common event in valvular disease. Walsh puts it second on his list, and in my 65 post-mortems I found it second. But as a single lesion it is extremely rare. I have found but one instance in my records, and there was not a single one in 252 cases of valvular disease that came to the autopsy table at the Massachusetts General Hospital. In fact, my search through the literature has revealed less than a dozen authenticated cases. These I have utilized for the purpose of framing rules for diagnosis, which are as follows:

(1) Acquired aortic obstruction occurs usually after middle life, and in males.

(2) The heart is large and the apex is carried down and to the left.

(3) The pulse is slow and hard and of moderate volume, until compensation has broken down.

(4) There is a systolic bruit up to the time the orifice has contracted to a mere slit.

(5) The murmur is best heard in the second right interspace, close to the sternum, and is carried up through the great vessels of the neck, being best heard on the right side.

In congenital cases, aortic obstruction, which is then a true stenosis, is only one of the numerous cardiac anomalies, the combination of which, as a rule, soon ends the infant's life. Very exceptionally patients may live with aortic stenosis of the congenital form to advanced life, according to Eshner,[1] who has reported one that lived to the age of 90 years. The duration of life, however, is said to be measured by the insufficiency that accompanies the obstruction or stenosis. I am not prepared to give an opinion on this question.

Gibson maintains that tricuspid insufficiency is the most common of valvular diseases; but Sperling makes the incidence only 26 per cent. in combined lesions, and less than 2 per cent. in single lesions. I have found the incidence 27 per cent. in combined lesions. Well pronounced tricuspid affections are rare; in fact, the most uncommon of all, if we except those of the pulmonary valve.

Tricuspid insufficiency is usually relative and due to many causes. But though it is comparatively rare, the number of recorded instances is large, for in Guy's Hospital alone 405 cases have been collected. I have not taken any trouble to add to them, as undoubtedly thousands could be found, all well authenticated. However, they afford a striking contrast to the recorded cases of tricuspid obstruction, which according to Herrick[2] had only reached 154 in the year 1897. And the relative variety is the usual one, Guy's Hospital records making it 97 per cent., while I make it 90 per cent.

Tricuspid insufficiency, however, is seldom recognized during life, even under favorable circumstances. In 29 cases proved by autopsies at the Massachusetts General Hospital but 5, or 17 per cent., were recognized during life. In 10 of my cases the diagnosis was made in only 3 (30 per cent.).

The reasons for this failure are owing partly to the difficulty in detecting the murmur (which it has even been said cannot be heard owing to the anatomic position of the valve and the course of the blood current), but also to the fact that even in hospital practice, where almost every heart murmur is studied at every seasonable opportunity, the tricuspid valve is thought to be of secondary importance as compared with the left-sided lesions with which it is apt to be associated. Thus it is apt not to be recognized except by those who are interested in every detail of cardiac pathology.

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[1] Proceedings of Path. Soc. of Phila., xiv, p. 154.

[2] Boston Med. and Surg. Jr., 1897.

(1) The most distinctive sign is the systolic murmur over a broad area which includes the lower half of the sternum and ensiform cartilage. The center of greatest intensity, according to my returns, is in the fourth left intercostal space close to the sternum, but sometimes as low as the fifth left intercostal space close to the sternum, and on the corresponding area of the right side, sometimes as much as an inch to the right of this point—so that the tricuspid area is somewhat broader on the right side than on the left. Occasionally this murmur may be carried to the right axilla, but it is seldom carried far to the left of the sternum. However, as the tricuspid area is large and rather indeterminate, as compared with the mitral, which is small and overlaps it, for purposes of differentiation, it is essential that the sound or bruit of the respective areas should be appreciably different, as to pitch, quality or duration, and time, while they should not be propagated in the same directions.

(2) The next most important sign is epigastric pulsation, but it may be absent.

(3) Another important criterion is the faintness of the second pulmonary sound, especially if previously it has been accentuated.

(4) The jugular pulse is very important, but not pathognomonic. Fulness of the jugulars is also an important sign, but to a less degree.

In exceptional cases the bruit of aortic stenosis in those very rare cases in which the bruit is carried from the aorta against the current down toward the apex may mislead, but the propagation of the systolic bruit up the great vessels in stenosis should prevent this error.

Tricuspid obstruction or stenosis is very uncommon. In 1881, Fenwick[1] had been able to collect only 46 cases. Pitt, in Allbutt's system, has collected 87, although Herrick, of Chicago, had previously collected 154. Since the latter period more cases have been published, so that according to my reckoning the present number is 162, not counting two of my own.

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[1] Trans. Path. Soc. of London, 1881, p. 46.

A feature of great diagnostic importance in tricuspid obstruction is that it is a woman's disease, par excellence.

Leubet, in his Thèse de Paris[1] (1888), found the ratio to be 4 to 1; Fenwick,[2] 8 to 1, and I 5 to 1 in the 12 cases that I have collected. There are signs in abundance, but usually they are insufficient for framing a positive diagnosis. The whole heart is moderately enlarged, but there is little, if any, enlargement of the right ventricle, although this auricle is usually dilated and sometimes hypertrophied. In one-half of the cases there is cyanosis, and it is apt to be extreme. There is also apt to be a pulsation, or at least a fulness of the jugulars. In 75 per cent., according to Colbeck,[3] there is albuminuria and dropsy. In 50 per cent. (Colbeck) there is epigastric pulsation and forcible heart action.

In 5 out of 7 cases, either a presystolic or a diastolic murmur was heard at intervals in the so-called tricuspid area, which according to English notions means over the fourth or fifth intercostal space to the left of the sternum. Others have put the presystolic bruit as occurring in 1 out of 8 cases. Some observers locate the greatest intensity of the murmur over the fifth or sixth right costal cartilage, or over the ensiform appendix, or over the lower half of the sternum, or as much as an inch to the left of it.

Thus far the diagnosis appears to have been made in only 6 of the 164 recorded instances of which I have records. These were 5 by Colbeck and 1 by Shattuck.[4] In about one-quarter of the cases no bruit was detected.

But it may be said in explanation that no thorough examination was made in a number of the cases because no opportunity was afforded, or attention had not been called to the tricuspid, or that the mitral obstructions that occurred in about one-quarter of them obscured the diagnosis, or, finally, that the auscultatory signs were fleeting.

In suspected tricuspid obstruction, the following points are to be remembered:

(1) Mitral obstruction in one-quarter of the cases accompanies tricuspid obstruction.

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[1] Quoted by Whitaker.

[2] Fenwick, Trans. Path. Soc. of London, 1881, p. 46.

[3] Medical Chronicle, 1893, p. 297.

[4] Boston Med. and Surg. Jr., vol. cxxiv, 1891.

(2) The right auricle is enlarged and dilated.

(3) There is a murmur in the diastolic interval in from 12 to 70 per cent., and it is best heard over the fourth or fifth intercostal space to the left of the sternum.

(4) There is palpitation, cyanosis, dyspnea, and edema.

(5) There is an epigastric pulse in about one-half the cases.

(6) In one-half of the cases there is a previous history of rheumatism. If, with these symptoms in a woman, there is a diastolic murmur in the tricuspid area, which in pitch, quality, and duration is different from the mitral murmur or sound, we may make the diagnosis of tricuspid obstruction with tolerable certainty.

Of all cardiac valves, the pulmonary is least often affected. Exceptionally, it is the single affection. Sperling did not find such an instance, however, but I have brief notes of one from the pathologic records of the Babies' Hospital of New York, with which I happen to be connected.

In combined affections, however, they occur in the ratio of 1 to 10 per cent. Sperling makes it 4 per cent., I make it 10 per cent., if I include mere thickening of the leaflets. But in a total of 175 valvular affections in my pathologic tables it occurred only 4 times.

All of these four cases of mine were examples of acquired pulmonary insufficiency.

Chronic insufficiency is not very common. Gerhardt,[1] in 1899, had only collected 29 cases verified by post-mortems, but Pitt in the same year had collected 99, of which 44 were taken from the pathologic records of Guy's Hospital.

The signs are:

(1) Displacement of the apex to the left. (2) In about 20 per cent. (Barié [2]) a diastolic thrill in the second or third left space, from the edge of the sternum to a point one inch to the left of it, and conducted down the left edge of the sternum. (3) In about 25 per cent. a double murmur. (4) The diastolic bruit intensified by inspiration. (5) Embolism of the lungs in one-third of the cases. (6) Murmur intensified by the sitting position. (7) Vesicular murmur, jerky after a long breath has been taken. (8) Hemoptysis. (9) Dyspnea. (10) Concomitant disease of the lungs,

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[1] Charité Annalen, xvii, p. 251.

[2] Barié, Arch. Gén. de Paris, 1891, vol. i, p. 650, and vol. ii, pages 30 and 183.

sepsis, or arteriosclerosis. (11) Usually hypertrophy of the left ventricle. (12) Epigastric pulsation. (13) In about 25 per cent. a systolic bruit. According to Barié, the diagnosis was made in only 13 of his 58 cases. On the other hand, in half of Pitt's septic cases the diagnosis was made, but an ulceration of the valve seems to be necessary to produce a murmur.

Pulmonary obstruction or stenosis is one of the most frequent of the many congenital cardiac anomalies that defy classification, owing to the number of varieties. It is generally recognized that the principal anomalies affect either the size or the position of the large vessels, the interauricular or interventricular septa, or the leaflets. The two most frequent occurrences are patency of the foramen ovale, or of the septum between the ventricles. But the persistence after birth of the ductus arteriosus, connecting the pulmonary artery with the descending aorta, is not uncommon.

Among the minor anomalies are strictures of the pulmonary artery or one of its branches, or narrowing of the infundibulum or conus arteriosus, lying beneath the pulmonary valves. But, after all, the danger to life and health is not proportionate to the number of defects, for one is apt to compensate another—a point which will be recognized without my going into details.

The literature contains an immense amount of material bearing on the matter of cardiac anomalies, and institutions caring for infants and children have many cases on record.

Pulmonary obstruction is rarely acquired. Constantine Paul,[1] in 1871, was the first to distinguish it from the congenital forms, but the ratio between the two has not been determined. However, congenital obstruction or stenosis is comparatively common. In fact, all of the cardiac anomalies are apt to be grouped about pulmonary stenosis, a point which is very helpful in hazarding a diagnosis. And the symptoms of the congenital form have some characteristics widely different from those of the acquired form.

In the congenital form there is usually cyanosis, though not always, as in a case in my records. But there is usually a lack of physical and mental development. The patient complains of headache, and is somnolent or has hebetude. He is also undersized, and has a bulging chest and a protruding abdomen. There is defective development of the genitals, and he has clubbed fingers and toes. The eyes may be prominent. The murmur is systolic and usually loud, if there are vegetations or erosions, and it may be heard all over the precordial area; but its intensity is apt to be greatest over the pulmonary area, and it is continued up to and sometimes under the clavicle. Exceptionally, it may be best heard lower down, and in one case (Hun's[1]) it was best heard over the fifth left sterno-clavicular articulation.

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[1] Soc. Méd. de Hôp. de Paris, tome viii.

In acquired obstruction or stenosis we should look for an antecedent infection, especially venereal diseases or rheumatism.

As distinguished from the congenital form, there is less often cyanosis, and there are none of the characteristics of arrested mental and physical development. If there is cyanosis, it is increased by coughing, and there is dyspnea. The murmur is more definitely located than in the congenital form, because the force of the blood stream is undiminished by defects in the walls of the heart. It may be loud and rasping, and should be so in the endocarditic cases. If the patient holds his breath, it should be fainter. The apex beat is apt to be forcible and diffuse, and there may be a thrill. The second pulmonary sound should be faint. But there may be insufficiency and a double murmur. Usually, there is an attendant pulmonary or bronchial disease of a purulent character.

The diagnosis is not easy. However, in the congenital forms, in which cardiac anomalies are apt to center around a stenotic pulmonary valve, it is comparatively safe to make the diagnosis of pulmonary stenosis, especially in the presence of cyanosis, in a person with arrested mental and physical development, even when the murmur is obscure or hard to locate; for a patent foramen ovale or a perforated ventricular septum will produce, the one a diastolic, and the other a systolic, murmur, and in infants it may be difficult to distinguish one from the other.

In the acquired cases, even after they have been examined in detail and frequently, the diagnosis has seldom been made. However, Constantine Paul and Mayer[2] have each recorded a successful antemortem diagnosis.

Pitt has collected 14 cases of acquired pulmonary stenosis, and

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[1] Hun, Albany Medical Annals, 1897, xviii, pages 57–66.

[2] Deut. Arch. f. klin. Med., Bd. xxiv, p. 435.

I have 2 in my records. Occasionally the stenosis may be temporary, as in one of my cases, in which a hepatic abscess appears to have temporarily compressed the heart, lungs, and great vessels, diminishing in particular the caliber of the pulmonary valve.

From the figures and statements that I have given, it is plain that there is still a pretty general lack of success in the diagnosis of valvular disease, even among the best clinicians. However, when distinctive signs are absent, in exceptional cases, when concomitant conditions are obscure, when the rules that have been accepted for guidance are misleading, or when the observer has not the proper equipment for making a diagnosis, failure may reasonably be anticipated.

On the other hand, there is every reason to believe that, with a larger experience in well-authenticated cases, we will reach greater accuracy in diagnosis. This fact is now being generally recognized, and efforts are being made, especially in this country and by our younger men, to frame newer and better rules based on the combined teaching of the bedside and the post-mortem table. And their efforts will react on hospital physicians and pathologists, making both more thorough in their work.

Indeed, our large hospitals are the sources to which we must look for the data to be utilized for our future guidance. And there is nothing more needed at the present time than a functionary whose special duty is to collect and utilize such statistics. The registrar of the English hospitals is supposed to perform this very important task. Who could be more helpful in the solution of many of the vexed questions of the day than a medical man well-equipped, trained, and balanced, with the hospital books before him? Such a man was at one time the late lamented Charles Hilton Fagge, of Guy's Hospital, London.

Surgery

THE DIAGNOSIS AND TREATMENT OF HEMORRHOIDS

A CLINICAL LECTURE IN THE COURSE ON SURGERY AT THE HARVARD MEDICAL SCHOOL

BY GEORGE W. GAY, A.M., M.D.
Senior Surgeon to the Boston City Hospital

Gentlemen: Last week you were shown some typical cases of enlarged (varicose) veins of the leg, upon one of which Dr. Bapst Blake had performed the radical operation of removing considerable portions of diseased veins with most admirable immediate results. You were also shown a case of dilated veins of the spermatic cord (varicocele), and you saw Dr. Blake remove about two inches of those vessels, and bring the stumps together with the end ligatures.

As was explained to you at the time, the object of this last procedure was to prevent the sagging of the testicle as much as possible.

If any operation for varicocele is necessary, this one is to be preferred. As a rule, none is required. In a large majority of cases a well-fitting suspensory bandage with ordinary hygienic attention in the form of cold sponging, freedom from constipation, etc., will suffice to keep the affection from increasing, until age completes the cure. That patient, a young man, was subjected to an operation for a specific reason, namely, to enable him to enter the public service.

To-day I am going to show you a case of enlarged and inflamed veins in a different locality,—that is, in the lower portion of the rectum. Hemorrhoids, or piles, are a common affection, one that will often tax your patience, your judgment, and your skill to manage satisfactorily to your patient and to yourselves. Occasionally an operation may be necessary, which most of you who undertake to earn your living by your profession will be called upon to do, and which you ought to be able to do successfully and well.

While our patient is being etherized and made ready for an examination and operation, allow me to say a few words to you upon the subject in general.

Piles are called *external* when they remain outside of the anus, the skin and mucous membrane in varying proportions forming their covering. *Internal* piles are located within or above the sphincters, and, of course, are covered solely by mucous membrane. Not infrequently the two varieties are found together, and if necessary may take both names, *interno-external* piles. This, however, is a refinement of nomenclature which is hardly worth our time.

Piles are most common in adult and advanced life. The young are less liable to be afflicted. Pregnant women are peculiarly subject to them for mechanical reasons. People who live high and exercise little are also frequent victims. The hemorrhoidal veins, having no valves and belonging principally to the portal system, are congested and distended by anything that interferes with that circulation. Hence the frequency of this affection in those having a sluggish liver and constipated bowels. Prolonged fatigue, exposure, horseback exercise, or a debauch may act as exciting causes in those individuals subject to the disease. In rare instances it is difficult to determine the cause of the attack.

The simplest form of external piles is the small, thin-walled, bluish tumor, a dilated vein, which may or may not be very sore, but it is always troublesome from being in the way in walking or in the toilet of the parts. Rest in a recumbent position and the application of cooling and astringent lotions will in time effect a cure, but the speediest relief is obtained by applying ice or some other benumbing agent for a few moments, and then slitting open the pile with a sharp, narrow bistoury, turning out the blood, and packing the cavity lightly with a bit of gauze or lint. The recovery is usually rapid, complete, and permanent.

We next have the so-called “fleshy pile,” which is simply a later stage of the former, and in which the venous coats are so much thickened that the tumors resemble tabs of flesh rather than enlarged veins. These tabs give little trouble except when inflamed. Then they may prevent walking, or even sitting with any comfort.

These external fleshy piles, like all others, may receive palliative or radical treatment according to circumstances. The former consists in rest in a recumbent position, the bed being the best place, and the application of soothing and astringent agents. Cold applications, as a small bag partially filled with cold water, agree with some patients better than hot fomentations or poultices. An ointment of extract of belladonna and glycerin, equal parts, or the ointment of galls and opium, or the dilute citrine ointment (1 part to 7) may be tried with or without the other applications. As is so often seen in our professional work, what agrees with one patient will irritate another; therefore, it is well to have a variety of remedies from which to make a selection.

If the tumors are pedunculated and have not too broad a base, removal with a scissors curved on the flat, is the surest and most permanent treatment. Care is requisite that not too much tissue be removed, or a stricture of the anus may be the result, thus leaving the patient much worse off than he was before. Only the most dependent portion of the tumors, say about two-thirds, should be cut off, trusting to cicatricial contraction to complete the cure.

Now we come to internal piles, which are covered entirely with mucous membrane and are located within or above the internal sphincter muscle. They may be in the form of folds of inflamed and thickened tissues and dilated veins, or they may exist as more or less distinct tumors. Occasionally one of the latter becomes fleshy and solid, and is then called a polyp. In almost all cases of hemorrhoids there is a certain amount of proctitis—that is, inflammation of the mucous membrane of the lower part of the rectum, and at times this condition is the chief source of trouble, in the way of pain, discharge of mucus, pus, blood, etc. All forms of piles may bleed, itch, give a sensation of fulness or bearing down, and when inflamed are very sore and painful.

To examine a patient for piles without ether, he should be directed to sit upon the stool for five to ten minutes and "strain down," as he would to move the bowels. He should then at once lie down on his left side upon a table with his knees drawn up as far as possible. With his right hand let him raise his right buttock, thereby giving the surgeon a good view of the ischiorectal region, at the same time leaving his hands free for any needed manipulations. By directing the patient to "strain down again" the parts will be made to protrude far enough to bring large piles into plain sight. Small ones will very likely require the use of the speculum for their detection. Fissures or ulcers at and near the anus will in this way be readily detected, and the lower portion of the rectum will usually be found to be red, inflamed, and more or less fissured with superficial ulcerations or abrasions. Small piles cannot be detected by the touch alone.

For a more thorough and satisfactory examination the patient should be etherized and placed in the lithotomy position,—that is, upon his back with his knees drawn up. There should be a good horizontal light; then with a Sims speculum the whole of the lower portion of the rectum can be thoroughly examined. If the sphincters are very tight, they should be stretched, as is always necessary prior to most operative procedures for internal piles. The rectum should always be examined as high up as possible with the finger to determine the presence or absence of growths or strictures. Malignant disease not infrequently begins three or four inches from the anus, and without a careful examination may easily be overlooked, to the detriment of the patient and physician. In most instances piles are located within two or three inches of the anus.

Treatment.—All cases of internal hemorrhoids do not require a radical operation. With care in diet, digestion, moving the bowels, etc., many of these patients get on fairly well without operation. Alcohol, rich or too hearty food, are to be omitted. Red meats, as beef and mutton, are to be taken sparingly. Poultry, fish, vegetables, fruit, especially if cooked, bread, and the cereals may be used freely; pastry and sweets very little. The bowels should be kept free with saline laxatives and injections. The mineral waters, as Hunyadi, Vichy, and a host of others nearly or quite as good, generally act very kindly in these cases. Calomel is apt to increase the irritation of an inflamed rectum, and hence should be used very sparingly in piles. Castor-oil is one of the very best remedies for certain cases of chronic diarrhea, but the ultimate effects are rather constipating, which precludes its use for any length of time in these cases. As a rule, the salines, either in the form of the salt or the natural waters, are the most satisfactory laxatives in piles. They should be taken on rising in the morning, are more effective taken hot, and can be used indefinitely. If too much is required to accomplish the desired effect, then an enema of warm water, with a little table salt or "saleratus," may be used to help along the laxative.

The old-fashioned mixture of equal parts of sulphur and cream of tartar taken in a little water acts very well in some cases. Fig syrup, Garfield tea, and a thousand and one different things are in use by the laity as laxatives, and many of them are not without merit in certain instances.

For acute inflammation of internal hemorrhoids the patient should remain in bed, take a light diet, and, if the pain is severe, should insert a small suppository containing one grain of opium or one-fourth of a grain of morphin every four to eight hours as required. Much relief may be obtained by small injections of either warm or cold water, as is most agreeable to each particular person. These injections are to be allowed to remain in the rectum indefinitely. Movements of the bowels are to be aided with copious enemas of warm water. The irritation following a stool may be relieved by a small injection of cold water, with or without a little hamamelis (one to four drams to a pint); it may be repeated as often as necessary. In many—in fact, in most—cases of irritable internal piles considerable benefit may be obtained from the judicious employment of suppositories. The one in most common use is probably the classical opium and gallic or tannic acid suppository, but I like the one used in the Pennsylvania Hospital much better. It is made up as follows:

℞	Lead acetate,			
	Tannic acid,	of each 15 grains	1	
	Lead carbonate,			
	Extract of stramonium,	of each 30 grains	2	
	Creosote,	5 drops		30

Cacao butter sufficient to make 50 suppositories.
Two to four of these are sufficient for ordinary cases.

I have used this preparation for many years with fairly satisfactory results. An ordinary "attack of piles," by which term is meant a more or less acute inflammation, will usually subside in from four to eight days if the patient take proper care of himself. The chief factor in the treatment is rest in the recumbent position.

Before touching upon the radical treatment of internal hemorrhoids I wish to draw your attention to a palliative method of considerable practical importance, but of which little is said in the books except in the way of disparagement. I have used the method occasionally for several years with very satisfactory results. I refer to the little operation of injecting piles with a ten per cent. solution of carbolic acid. This method is applicable only to those tumors situated above the sphincters, or those that will remain there when so replaced. As injected piles swell considerably for a time, the external variety should on no account receive this treatment. They will be made much worse, and no ultimate benefit need be expected from it.

The solution is composed of carbolic acid (ninety-five per cent.) one part, glycerin and water, each five parts. One, or, at the most, two drops, according to the size of the pile, is to be injected into the center of each tumor with an ordinary sterilized hypodermic syringe. The operation may be done in your office in the following manner: The patient is directed to sit on the stool and strain down, as he would to empty the bowels, for ten minutes. He is then to lie down on his left side and raise his right buttock with his right hand. By straining down again the piles will usually present at the anus and can be readily injected. It is sufficient to inject two tumors at one time, and the operation may be repeated in about a week on other tumors. It is seldom necessary to inject a tumor a second time inside of several weeks, or even months. Large tumors may require two drops of the solution, but usually one is sufficient at each injection. Little or no pain attends or follows the operation, and the patient goes about his usual business at once. No special precautions are necessary by reason of the operation, and the relief to the hemorrhage and enlargement of the veins is speedy and more or less prolonged. This is not a radical cure for piles, but in large or bleeding piles the relief is pretty certain, so much so that some patients prefer this method to one which is expected to bring about a permanent cure. The precautions to be observed are the following: Inject only *internal* piles; the strength of the acid solution should not exceed 10 per cent.; not over two drops should be injected into each pile; the operation should not be repeated inside a week; promise relief only, and not a permanent cure, as a result of the treatment.

The more radical measures resorted to for the relief of internal hemorrhoids are nitric acid, ligation, and clamp and cautery. The acid treatment is adapted for the red, velvety, thickened condition of the lower portion of the rectum, but not for distinct tumors. Abrasions and fissures giving rise to pain, bleeding, itching, and so forth, are benefited by the acid. The application is made as follows: The patient having been etherized and placed in the lithotomy position, the sphincters are to be thoroughly stretched until they are felt to give way or become completely relaxed. This is done to prevent the suffering which is pretty sure to arise from the spasmodic contractions of those muscles during the subsequent twenty-four hours, and should precede all operations for the radical cure of piles. A Sims speculum and a good horizontal light will enable you thoroughly to see and examine four or five inches of the lower part of the rectum. You can thus detect the condition of the mucous membrane, as well as the presence of ulcers, piles, or whatever may be abnormal.

The diseased tissues are now to be lightly touched with a glass rod or simple stick dipped in pure nitric acid. This turns the mucous membrane white, and in a few moments the rectum should be douched with a saturated solution of bicarbonate of soda to neutralize any free acid which may be present. The action of the acid is limited to the surface, and does not affect the deeper parts, as would caustic potash. A suppository containing a grain of opium or one-fourth of a grain of morphin is inserted high up in the rectum; a firm pad of sterile gauze is applied to the anus and secured by a T-bandage. The pain following this operation is of only moderate severity and is easily controlled by an occasional suppository. The ultimate results are usually marked and satisfactory. The acid may also be used in connection with the ligature.

Piles are tied in the following manner: A tumor is seized with volsellum or other forceps and dissected up from below, leaving a moderate pedicle above containing the vessels. One ligature is sufficient to completely strangulate a pile not larger than the end of the finger, and is to be tied tightly around its base. Larger tumors are to be transfixed with a needle carrying a double ligature, and the pile is to be tied off in sections. The ligatures must be drawn tightly and secured by three square knots instead of two. The ends are to be cut short, and any redundant portions of the pile may be trimmed off, care being taken to leave sufficient pedicle to prevent the string from slipping. All the tumors having been tied in this way, the after-treatment is similar to that following the other methods. The ligatures take care of themselves and come away in about a week. The ulcerated surfaces, the site of the piles, require several weeks in healing, but the most of the trouble is over in two or three weeks.

After that time it is generally safe to allow the patient gradually to resume his ordinary vocation.

Finally we have the clamp and cautery operation, which is the favorite method of removing internal hemorrhoids with most surgeons of the present day. Convalescence seems to be of rather shorter duration by this plan than by ligature. That the ultimate result is any better I very much doubt. The preliminary steps are to be carried out as in the previous operation. The sphincters having been thoroughly stretched, a pile is seized with forceps, and a stout clamp applied firmly at its base. Unless the tumor is too large, the whole of it may be charred and burnt off with a Paquelin cautery at a dull red heat. The pedicle should be slowly but thoroughly charred to seal up the vessels and prevent hemorrhage. Care is necessary that the clamp does not become hot enough to do harm. On loosening the clamp there should be only a slight oozing, which will soon stop without special treatment. Should it not do so, the spot may be lightly seared with the cautery. Each tumor is to be treated in this manner, the after-treatment being the same as in the previous operation.

Care is to be taken that no skin be burned or included in the ligature, as needless pain is pretty sure to result therefrom. Retention of urine is liable to follow any operation for piles of any severity. To avoid the danger of an infectious cystitis I should advise you to delay the use of the catheter as long as possible, for twelve, eighteen, or even twenty-four hours in some instances. There is little risk in allowing the patient to get up and sit on the closet to pass water if he cannot do it lying down. After operation by ligature or clamp and cautery confinement in bed and a light diet are necessary for two or three days, when the bowels may be moved by Hunyadi water or castor-oil, best taken in capsules. Solid food in moderation can then be allowed, but the recumbent position, mostly in bed, had better be continued for about two weeks, in order that the tissues may be well advanced in the healing process before permanently resuming the upright position. Generally speaking, the convalescence following these operations lasts from three to six weeks. The ultimate results are usually permanent and satisfactory after operation by clamp and cautery or the ligature.

Carefully done under aseptic precautions, very little danger attends operations for piles in fairly healthy people. There is always an element of danger in operating upon old people, the dissipated, the weak and delicate who have low vitality and low resisting power which must be carefully considered. It is for you to decide in each case whether the conditions are such that you will be justified in resorting to radical measures, or had better confine your efforts to safer, even if less effectual, methods. To decide these questions calls for good judgment, one of the very best attributes of the good physician, and one that should be cultivated to the utmost extent of your abilities and opportunities. Before resorting to any operation for any affection you should study the case from all sides. Carefully consider the probable results without an operation as well as with one. Always base your decision upon your judgment rather than upon your impulse. In fact, a surgeon should have no impulses, for unless they are under efficient control they are continually leading him astray. Beware of making diagnoses and doing operations upon the spur of the moment. They are more apt to be wrong than right. It is far better to be safe than sorry. (A patient was operated upon for internal hemorrhoids, and the various methods for a radical cure were demonstrated.)

ABDOMINO-PELVIC DIAGNOSIS: ABDOMINAL SWELLINGS

BEING LECTURE II OF A SERIES OF LECTURES ON ABDOMINAL DIAGNOSIS[1]

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GENTLEMEN: The second primary complaint of patients suffering from abdomino-pelvic disorders is *swelling*. The subjects so affected form a very large class, which it is necessary to subdivide. The most ready way is to consider those swellings which are directly visible on inspection and those which require bimanual examination in order to distinguish them. The first group immediately divides again into those conditions which are seated in the abdominal wall itself and those which lie internal to this.

I

The best method of separating swellings which arise in the wall from those beneath it is that given by Jenner. All such swellings are made more prominent by setting up contractions in its muscular layer. The muscles of the abdominal wall lie deeply. There is nothing between them and the peritoneal cavity but the extremely thin layer of parietal peritoneum, and usually almost as thin a layer of subperitoneal fat. This layer is badly named, since it actually lies above the peritoneum, and should rather be called the submuscular, or supraperitoneal layer. However, the term "subperitoneal" is universally used, and as long as the real position of the tissue is remembered, its name is not of much consequence. Above the muscular layer lie the deep fascia, the subcutaneous fat, which is often an inch or more thick, and the skin. All mural swellings lie in or above the muscular layer, which, if it contracts, forces them forward. The test therefore depends on this arrangement of layers.

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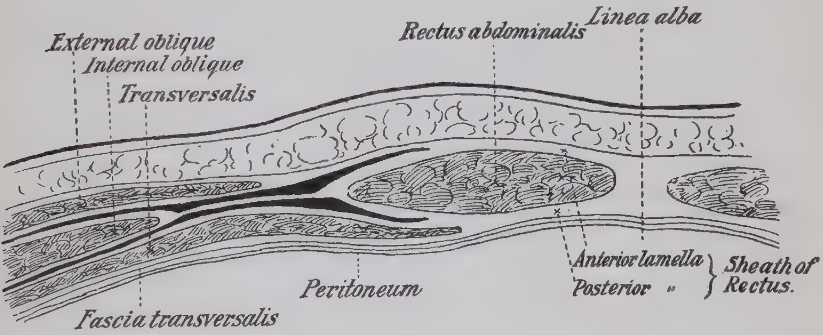
[1] Lecture I, in which the diagnostic significance of pain was discussed, was published in the INTERNATIONAL CLINICS, Twelfth Series, vol. iv, p. 63.

A word in passing as to the deep fascia. Its name also is misleading. We come upon deep fascia elsewhere, as, for instance, in the thigh, where the term represents a firm fibrous sheath covering in and isolating the muscles beneath, but having no direct connection with them. In the abdominal wall things are different. The deep fascia of the abdomen is really a broad tendon, the direct continuation of the muscles on either side. This is shown very well by these figures representing transverse sections of the abdominal wall, taken from Braune's photographs. (Figs. 1 and 2.) You will see that the three lateral muscles—transversalis, external oblique, and internal oblique—end in tendons at Langenbuch's line, just outside the vertical recti muscles, and that all these tendons



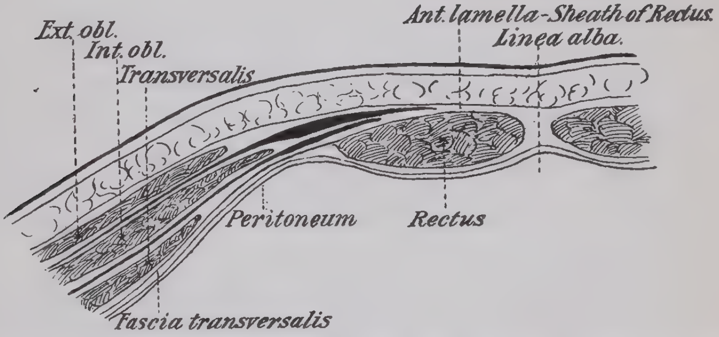


FIG. 1.—Transverse section of the anterior abdominal wall above the fold of Douglas, showing the formation of the deep fascia.

FIG. 2.—Transverse section of the anterior abdominal wall below the fold of Douglas, showing the formation of the deep fascia. (After Braune.)

join to form a thin broad tendinous layer, which, above the fold of Douglas, splits to enclose the rectus; but below that position it passes entirely in front of that muscle to unite with its fellow of the opposite side in the middle line, forming what is known as the linea alba. The linea alba is really the junction of these tendons in the central line, and this combined tendinous expansion is known as the deep fascia. It is important to remember this, for two reasons: (1) Subperitoneal fat sometimes comes through some small opening in this fascia. There are only two lines where this is possible—at Langenbuch's line and at the linea alba; and this is a great help in diagnosing them. At all other points there is too great a bulk of muscle between the subperitoneal fat and the skin for such hernias to occur. (2) Ventral hernia, which usually is a result of operation in which these tendons have been divided and not reunited with sufficient care, is always likely to increase progressively, since the muscles on either side, when they contract, pull steadily on its periphery, their action not being neutralized, but now increased by the pull of the opposing muscles on the opposite side.

The Jenner test referred to is this: If the patient lies on his back and attempts to rise into the sitting posture without the help of his hands, he must put the abdominal muscles into a state of contraction, since they are the only muscles which in this position can lift the thorax. Many people cannot actually lift themselves in this way, but this does not matter. The effort to do so involves contraction of the abdominal muscles, and a swelling situated in or above them is thereby rendered more prominent. A swelling situated beneath them, on the other hand, becomes less prominent and less distinct, on account of the resistance so produced, so that we have a ready way of differentiating the two—the reason for which is evident. You will see that in every case this must be so; there can be, therefore, no exceptions.

Having decided, by the foregoing test, that the swelling is in the wall itself, we have first to eliminate hernia.

You will see that the essential difference between a hernia and a true swelling of the abdominal wall is that the hernia, being a temporary displacement of organs originally formed in the abdominal cavity itself, must have an organic connection between the protrusion and the parts beneath. No posterior definite line of demarcation, therefore, can be felt. The fingers of the examiner,

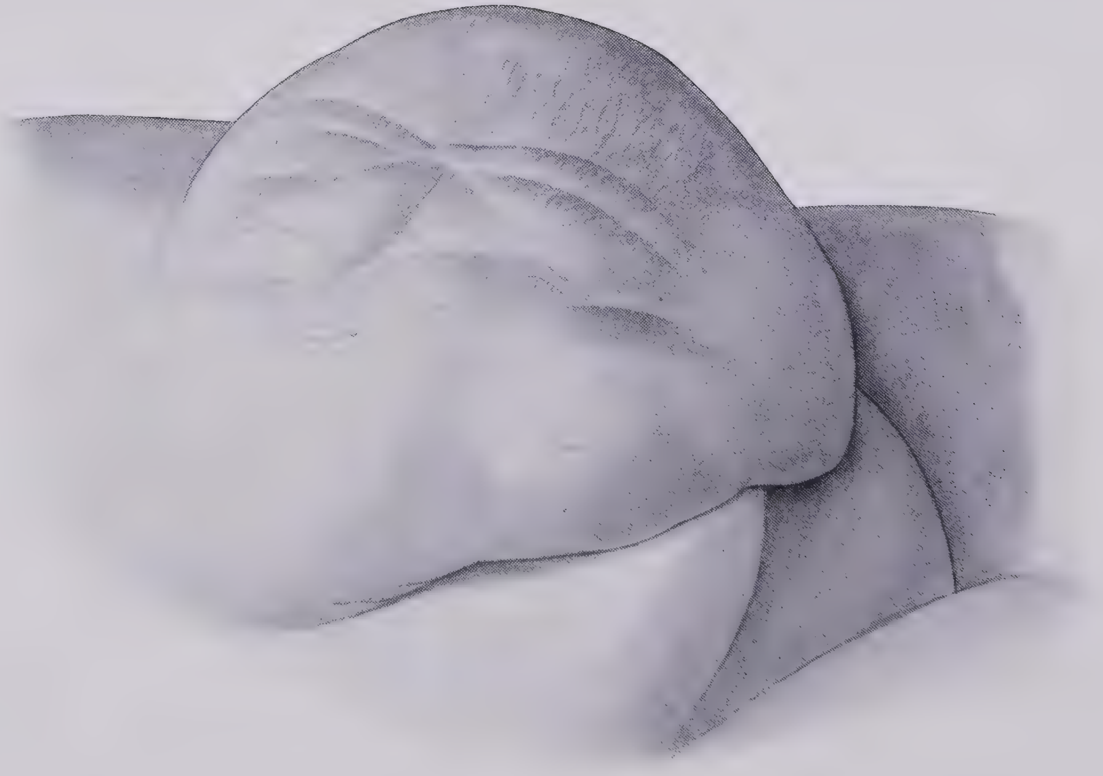


FIG. 3.—Large umbilical hernia.

however deeply placed, can never get actually behind it. This is not the case with a true swelling of the wall itself. This gives us our distinguishing characteristic.

Ordinary hernias occur at certain well-known and definite points: the umbilicus, the two external inguinal rings, and the femoral rings. I do not mention diaphragmatic or obturator hernias, because these do not form swellings in the abdominal wall. *Umbilical hernia* can scarcely be mistaken for anything else. Here is an exceptionally large specimen; but even this is unmistakable. (Fig. 3.)

Inguinal and femoral or crural hernias are sometimes extremely alike. A femoral hernia, when once it has attained a certain size, climbs upward into the inguinal region, so that at first sight it appears to occupy exactly the same position as one which has passed through the inguinal ring; but they can be distinguished by one thing: *Find the pubic spine. As this lies below and outside the inguinal canal,* it must be *below and outside an inguinal hernia* which passes through that canal; as, on the other hand, it is *inside the femoral opening,* it must be also *inside a femoral hernia.* I need scarcely say that it is of the first importance to differentiate these two, since, in attempting to reduce such hernias, pressure must be employed in directly opposite directions. In inguinal hernia you press upward and outward; in femoral hernia pressure must be applied at first downward and inward, then backward and upward.

Of hernia occurring elsewhere than in the usual positions, there are three which must be remembered.

Ventral hernias have almost always a history of some operation on the abdominal wall; therefore there will be a scar near or over them. They are irregular in shape and easily reduced, as a rule; and when reduction has been effected, a wide, thin-edged opening, through which they have escaped, can be felt in the musculo-tendinous wall beneath the skin. They return with equal facility on removal of the reducing pressure.

Lumbar hernia is rare. When it occurs, it is found just above the iliac crest at the side and at the junction of its posterior with its middle third. The protruding intestine comes through the canal of Petit, which is really a triangular gap between the internal oblique muscle in front, the latissimus dorsi behind, and the iliac crest below. This gap is not always or indeed often present, as usually the latissimus and oblique muscles overlap each other at their edges and the areas of their insertion into the iliac crest. In such a case hernia here would be impossible. When it occurs, a swelling is formed at this point, which, if it increases, tends to do so in an upward direction.

Pudendal hernia occurs, of course, only in women. It is rare, but when it occurs it may be easily mistaken for other things. The bowel in this case protrudes in the vagina, pushing the vaginal mucous membrane before it. It passes out of the pelvic cavity below the pubic ramus, beneath the triangular ligament. It therefore *lies on one side of the vaginal wall,* and if it comes far enough forward, *bulges out one of the labia majora.* It feels like a sausage, pushed end forward, the anterior extremity being rounded and well defined, whilst its posterior extremity passes beyond reach into the pelvis, and has no definite outline. It may be mistaken for cystocele, rectocele, a cyst of the vagina, or an abscess or growth in Bartholini's gland. From the first two it differs in that it is markedly lateral, neither anterior nor posterior, but between the two. The opposite side is easily contrasted with the side affected. Cystocele or rectocele affect both sides equally. In each of the three other conditions the posterior outline is as well, if not still more defined than is the anterior outline—the finger can be passed behind them and their limits are easily palpable. *This cannot be done with pudendal hernia.* Pressure usually will reduce it directly backward and upward.

But there is one form of hernia that may occur which is not intestinal, and when it does it may cause hesitation in diagnosis. This is *hernia of subperitoneal fat.* A small nodule, the size of a pea, or less, may be found in the linea alba, or in one or other of Langenbuch's lines, very tense, irreducible, and to a certain extent movable in the tissues around it. It must occur in one of these three lines, because elsewhere the muscular layer, as I have already said, is too thick for it to be possible, or rather, one should say, that it might occur elsewhere, but such an occurrence would be very improbable, and it could not then be discovered. If it can be felt at all, it will be in one of these lines.

For what might it be mistaken? For a small neuroma; a small ventral hernia, especially of omentum; a fibroma; a lipoma; or a small cyst.

A *neuroma* may be excluded by the fact that it is painless. Neuromas are always painful on pressure, often without. This little hernia is not painful on pressure. A small *ventral omental hernia* can be differentiated by the sensation on palpation. Omentum has a very characteristic granular feel. This is firm, though soft, smooth, rounded, not granular at all. The opening also by which it escapes is usually minute, whilst an omental hernia has relatively a much larger space behind it. From a minute *fibroma* it is separated by its soft consistence. A fibroma, however small, is much harder and more compact. A small *cyst* is so extremely rare in the abdominal wall itself that the possibility of its occurrence may almost be ignored. As it is practically a lipoma itself, this condition is naturally the most difficult to exclude. But true lipomas are either diffused or encapsuled. The former could not be mistaken for it, the latter might. The only clue for distinguishing them is by considering their origin. An encapsuled lipoma is formed in the layer of the abdominal wall in which it is found. Hernia of subperitoneal fat has escaped from one layer into another, but it is still connected with its original site by a pedicle. Now, any growth with a pedicle can only move as far as its pedicle will permit, and in this case the pedicle is very short. *The growth is therefore anchored to one spot.* Encapsuled lipomas, on the other hand, are not bound; they are capable of some movement, and *especially can be lifted away from the deeper structures* with comparative freedom. In this way distinction is fairly easy.

Having satisfied ourselves that the tumor observed is not a hernia, we arrive at its diagnosis by certain definite steps.

All other tumors of the abdominal wall may be sharply divided into two classes: (1) Those which infiltrate the wall around, and (2) those which have a definite limiting outline. Of the first class we have acute and chronic abscess, syphiloma, sarcoma, actinomycosis, and carcinoma. Of the second class we have nine: Glandular enlargements, fibroma, lipoma, ectopia vesicæ, phantom tumor, papilloma, condyloma, and Bartholini's and sebaceous cysts.

(1) We may divide *swellings which infiltrate the tissues around,* and have therefore no definite circumscribing line, into those which are associated with a definite rise of temperature and those which have none. Of the first subclass there are three: Actinomycosis, acute and chronic abscess. *Acute abscess* is easily

distinguished from the other two by the more rapid and steady rise of temperature, by the greater tenderness, the acute throbbing pain, and the expression of suffering on the patient's face — none of which are seen in the other two. *Chronic abscess* and *actinomycosis* are in their earlier stages almost indistinguishable. Both have the same irregular and not well-marked elevation of the temperature; there is the same boggy swelling, with a reddened, shiny surface, and the same slow increase, although actinomycosis is perhaps the more sluggish of the two. Later there is a greater tendency to burrow and to open in various points in actinomycosis than in simple chronic abscess, which bursts at one point, its center, if it bursts at all; the pus of a simple chronic abscess is thick, creamy yellow, whilst that of actinomycosis is thin and watery, with minute greenish specks floating in it. An absolute diagnosis of the latter, however, must depend upon the discovery in its secretion of the special fungus, the growth of which is its cause. Actinomycosis is also rare in this situation. It is far more often seen in the jaw, tongue, pharynx, or in some internal organ.

About the other three, syphiloma, sarcoma, and carcinoma, it is sometimes more difficult to form a certain opinion, but *carcinoma,* when it occurs in the abdominal wall, usually attacks the umbilicus, and is secondary to hepatic cancer. A hard, nodular, bossed edge of the liver can therefore usually be detected by careful palpation in such cases. The growth itself is nodular and hard. Any ulcer formed, and it ulcerates fairly soon, has similar hard, everted, nodular edges.

But *syphiloma* and *sarcoma* are very much alike in appearance—at all events at first. There is the same swelling infiltrating parts around or — for sarcoma is sometimes encapsuled and a gumma is sometimes fairly defined—the same isolated lump, covered by unchanged skin, growing with fair rapidity. Should the swelling be limited on one side, whilst infiltrating on the other, that is in favor of a syphilitic cause. In such cases the history of injury on the one hand, of specific infection on the other, may decide the question. A patient's statements as to the possibility of syphilitic infection are not always to be believed. It may when present be totally denied, or, on the other hand, admitted, when the fact is that nothing more than gonorrhea or a harmless balanitis or simple urethritis has preceded it. Such facts as previous rashes on the chest, the palms of the hands, the flexor surfaces of the limbs, or the forehead, alopecia, repeated miscarriages in the female, prolonged sore throat in either sex, are then of great significance as tending to prove previous syphilitic infection, or their absence to disprove its existence. And failing certainty in this direction, it is always wise to try the effect of a short but effective dosing with iodid of potassium along with Plummer's pill (calomel and sulphurated antimony, of each, one-half grain—0.03 gram). If this brings about rapid diminution in the swellings, their syphilitic origin may be pretty certainly considered as proved. It is well to note that this is not given as a point of difference between syphiloma and actinomycosis, as the latter is almost equally affected by this drug.

(2) *Swellings having a definite limiting outline.* One of these can be immediately eliminated; all the rest are covered by skin. This alone is *covered by mucous membrane,* the exposed lining membrane of the bladder. It can be nothing else than *ectopia vesicæ.* The mucous covering is a pathognomonic sign. So also is the escape of urine in small rhythmically occurring spurts from two points in the upper portion of the mucous area. Confirmatory signs are the presence of a small, badly developed penis, of a separation between the pubic rami, and of excoriations of the skin around.

The remaining swellings may be divided according to their position. *Papilloma* and *condyloma* lie above the skin line, are implanted upon it; all the rest, enlarged glands, lipoma, fibroma, phantom tumor, sebaceous and Bartholini's cysts, lie beneath the skin.

We distinguish *papilloma* and *condyloma* first by their position. Condylomas lie around some natural opening, the anus, vulva, or urethra; *papillomas* may be found on any portion of the skin. Condylomas are moist, flattened; papillomas are dry and pointed. Both may be dendritic; that is, from a central stem or pedicle processes may grow outward at varying angles. Papillomas occur without any very obvious cause; condylomas are usually the result of, and are associated with, some irritating discharge from the canal around which they are placed.

Of swellings below the skin and having a defined outline, *Bartholini's cysts* can only occur in women, and in the lower half of one or both labia majora. They are elastic, fluctuant, and sometimes semitranslucent.

Phantom tumors are due to spasmodic contractions of muscular fibers, usually of segments of the rectus muscle. They are, therefore, usually met with on either side of the middle line and in the upper segment of the abdominal wall, *i.e.,* above the umbilicus. Their pathognomonic symptom is the fact of *their disappearance under chloroform,* which annuls the spasm. No other swelling is so affected by an anesthetic.

Four other swellings remain, enlarged glands, lipoma, fibroma, and sebaceous cysts.

Sebaceous cysts are really formed in the skin itself, so that the skin is not movable over them. It is readily movable over the other three.

Enlarged glands are only seen in the inguinal region. They are very rarely single, but occur in chains, the members of which are distinct. They lie close above the line of flexure of the thigh, are movable to a slight extent upward and downward, but much less in a lateral direction, owing to the thickened lymphatic vessels which connect them. They cannot be lifted away from the deep fascia upon which they rest. They are rounded, firm, and vary in size. They are usually secondary to some irritation about the skin of the penis, scrotum, vulva, perineum, or buttocks, or to some irritating discharge from the vulva or penis, as in gonorrhea, soft or hard chancre. Their position, number, and secondary character distinguish them from lipoma or fibroma. When inflamed, their characters alter to those of abscess (*q.v.*).

Lipoma and *fibroma* differ essentially, in that the former is soft, the latter hard. Confirmatory signs are: Fibroma is either a single, hard, rounded mass, or a congeries of such masses so connected as to form a multilobed hard mass. Its edges cannot be rolled under the finger. The skin over it does not dimple if stretched or pinched up. Lipoma is a single, soft, rounded, but more flattened mass, or a congeries of such masses so connected as to form a multilobed soft mass. Its edge can be rolled under the finger. The skin over it dimples if stretched or pinched up.

So much for swellings occurring in the abdominal wall itself. It will be noticed that all such swellings represent structures normally present in the tissues composing that wall, abnormally developed, or they are syphilitic or malignant growths.

II

As to swellings which lie below the anterior abdominal wall, they are so many, and so various, that it is necessary to subdivide them in several ways in order to deduce any information as to their character.

Having satisfied ourselves by Jenner's test that the swelling in question does not lie in the wall itself, but beneath it, we proceed to decide whether it is *common to the entire abdominal cavity or limited to some portion of it.* The abdominal cavity itself may contain gas or fluid of some kind. If gas, some portion of its wall naturally dull will give a clear note on percussion, or its areas of normal dulness will be lessened. Such areas are those normally occupied by the liver or spleen, and the former being the most marked is usually chosen for the test. If fluid, this will naturally gravitate to that portion of the cavity which lies the lowest in any given position of the body; and this area will naturally be different in different positions. Thus, whilst the patient lies supine, the lowest area available for percussion will be the flanks—the renal fossas; on raising the body to the sitting position, these regions will become clear, whilst the hypogastrium, previously clear, will become dull. The rapidity with which these changes appear will at the same time suggest the consistence of the fluid present: whether blood, serum, gastric, or intestinal contents, or colloid material.

If the swelling, on the other hand, is limited by a definite boundary line, we have to distinguish between those springing from an *abdominal organ* and those which have a *pelvic origin.* Here another test, also introduced, I believe, by Jenner, will give us the means of distinguishing them. The patient is placed in the dorsal position; the shoulders and knees are raised and supported by pillows, so as to relax as much as possible the abdominal wall. The examiner's hand is placed on the abdomen just below the transverse line between the anterior superior iliac spines. With each respiration the hand sinks lower and lower toward the sacral promontory. If this or the aortic or common iliac pulsation can be recognized below the swelling, such swelling must arise from an abdominal organ. If none of these can be felt, it is almost as certainly pelvic in origin. We shall in this lecture only be able to go somewhat cursorily over the first class. The second class must be dealt with in a future lecture, in which the bimanual method of examination which is most suited for such conditions as are there to be found will also be discussed.

Taking, then, swellings having an abdominal origin, the next step is to determine the nature of their contents: whether *solid* or *fluid*. In settling this point, puncture is never justifiable. Paracentesis abdominis is becoming year by year less and less required. Only in ascites is it permissible; and even here the advantages to be obtained by Morrison's operation of laparotomy, combined with union of the omentum to the abdominal wall, in preventing refilling and ameliorating the results of backward pressure in the portal vein, render this blinder method of relief less advisable. But in any case in which the contents of an abdominal swelling are plainly encysted, paracentesis is not merely obsolete; it is almost criminal. If the tumor is solid, the veins contained in it may be wounded, and bleed afterward into the peritoneal cavity to a dangerous extent. If it contains fluid, some of this will almost certainly continue to flow after the removal of the cannula, not through the opening in the skin, which is at once displaced, but into the cœlom, and this fluid may be infective in various ways. The dangers of the practice cannot be too strongly insisted upon. The character—fluid or solid—can usually be easily determined by the trained hand. Palpation for this purpose should be gentle and most patient, the tumor itself being either supported by the examiner's other hand or pressed against some resistant portion, as the spine of the patient's own body.

We have next to ascertain the *mobility or immobility* of the circumscribed swelling. All post-peritoneal—except renal—swellings are *absolutely immobile.* Of course, in speaking of post-peritoneal or intraperitoneal swellings one is speaking clinically. From a purely anatomical or embryological stand-point every structure is post-peritoneal. Such immovable post-peritoneal tumors, if not aneurismal or pancreatic, are syphilitic or sarcomatous neoplasms. Renal tumors must, however, be considered exceptions; these, although post-peritoneal, are movable to some extent. Retroperitoneal glands, although enlarged, are seldom sufficiently so as to be capable of palpation, still less of inspection, without laparotomy;

whilst, although a calculus in the mid-course of a ureter might theoretically be considered capable of being palpated, I have never been able to do this until it has reached the pelvis, nor am I aware that it has ever been certainly done.

On the other hand, all intraperitoneal swellings have some degree of mobility; and this mobility may be of use in diagnosis in various ways. In the first place, certain swellings move synchronously with the movements of respiration. Those which move easily must be in close connection with the diaphragm; thus hepatic and gall-bladder swellings move most of all. Not only do they move downward with deep inspiration, but they move upward again with expiration, and cannot be prevented from so doing by the examiner's hands. The same is true of swellings involving the upper part and body of the stomach, but not of those connected with the pylorus. Splenic and renal swellings, not being so intimately connected with the diaphragm, move downward with forced inspiration, but move upward again somewhat sluggishly, or not at all, and can often be held by the examiner's hand in the position which inspiration has given them.

Certain swellings have very free movement and are even acted upon by the force of gravity alone. If the patient is placed in the erect position, the tumor which when he was lying supine occupied the central or epigastric region may now be found in the hypogastrium, or in one or other of the iliac regions; whilst if the foot of the bed be raised sufficiently, or still more, if the patient be placed in the Trendelenburg or knee-chest position, the swelling will be found to have migrated to a new location beneath the ribs on either side. Such complete mobility can only be found in a subperitoneal fibroma or ovarian cyst, the pedicle of which has yielded —and such are rare—or in omental, mesenteric, or small intestinal swellings.

Without any of this exaggerated mobility, tumors capable of movement either by the examiner's hand or by changes in the position of the body are of frequent occurrence. Inasmuch as any moderately mobile tumor must have, if only for the entrance of vessels, some connection with a more fixed part, information as to its point of origin, which will be that of this blood-supply, will be obtained if the area through which such movement takes place is carefully noted. A given point on the surface of the tumor farthest from the presumed attachment must during its movement describe an arc, which will be part of a circle the center of which must lie at the spot at which these vessels enter. This spot can thus be determined, and the organ which normally occupies such a location will almost certainly be found to be that from which the growth has arisen. The organ once identified, the kinds of growth peculiar to it may be passed in review and their differentiation effected by other observations.

Swellings of the abdomen may not only move with respiration or be capable of movement by the examiner's hand; they may possess independent movement. This is, of course, most marked in those connected with the digestive tract; and this movement may be very slow, or sufficiently fast at once to attract attention. In most cases of acute obstruction the small intestine becomes dilated and distends the abdominal wall. Peristaltic wave-like movements may then be easily watched, causing the abdominal wall to rise gradually and fall again as if a ripple were passing over its surface. But it is in chronic obstruction, and more especially in that affecting some portion of the large intestine, that such movements are best seen. In a case of stricture of the sigmoid flexure pretty low down this was very well seen; the whole large intestine was distended, and the sigmoid itself formed a large coil in the hypogastrium, the movements of which could be easily watched through the emaciated parietes. In such cases the layer of subcutaneous fat is often much decreased in thickness, and the abdominal wall itself is thinner and more lax than usual.

In dilatation of the stomach contractions of this viscus can sometimes be seen, especially if it is distended by gas or fluid. A rounded swelling emerges from beneath the left costal margin and passes downward and to the right in a more or less oblique direction. In acute intussusception, during a spasm of pain, a swelling may apparently develop, and as the pain subsides, fade away and disappear. In chronic intussusception, whilst no appreciable alteration in position may occur during any one observation, the tumor will be found to have materially altered its site if successive observations are carefully recorded for a few days. The same unobtrusive movement may be noted in swellings due to tuberculous peritonitis. Fecal masses supply perhaps the most startling changes of site, however. At the first examination they may appear to be as hard and fixed as carcinoma,—although often doughy in consistence, it is a mistake to expect them always to be so,—the next day they may have entirely disappeared, or occupy a perfectly new situation.

The *outline of the abdomen* viewed from the side,—the examiner's eye being on the same level,—the sky line, so to speak, will often give information. Kelly has drawn attention to this point. The movements of respiration affect it. In a normal abdomen the sky line moves synchronously with inspiration and respiration, rising with the former and sinking with the latter throughout its entire length. Should any sufficiently resistant tumor, however, be present, its movement is checked at the point where the mass touches the anterior wall, just as a violin string ceases to vibrate at the bridge, or when pressed upon by the player's finger.

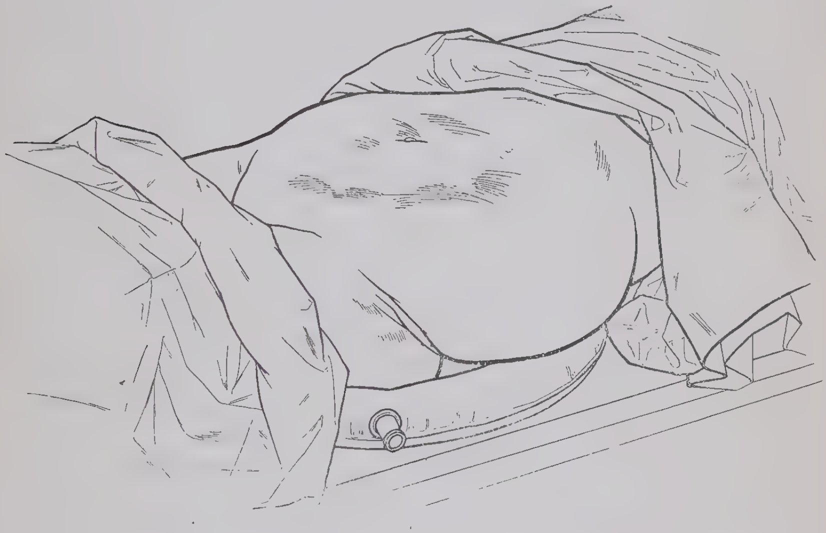


FIG. 4.—Outline of abdomen showing the condition in ascites when not fully distended. (After Kelly.)

Certain swellings have almost characteristic sky lines. Thus the swelling produced by ascites, if the abdomen is not fully distended, is flattened in the center whilst bulging in the renal regions, and the sky line will dip in the center. (Fig. 4.) If the distention by fluid is great, the whole sky line is convex equally from the xiphoid cartilage to pubes; the abdomen projects as a whole. The swelling of a pregnant uterus also projects prominently forward in the center, but the sky line is flattened above near the xiphoid, bulging out firmly an inch or two below.

The manner in which the sky line ends is also to be noted. In pregnancy this line rises gradually from the pubes, but dips abruptly near the xiphoid. In uterine fibroma the dip is nearly as abrupt toward the pubic as the xiphoid extremity. There are, however, two main forms in this disease, both with marked rise above the pubes; in one the outline above this rise is a regular curve, as if over a large round ball; in the other, irregular, as if covering several differently sized mounds. (Fig. 5.) The rounded, even form may, however, be distinguished from a similar appearance due to ovarian cystoma by the fact that whilst in both the rise from the pubes is abrupt, that from the xiphoid end is abrupt also in fibroids, but gradual in ovarian disease.

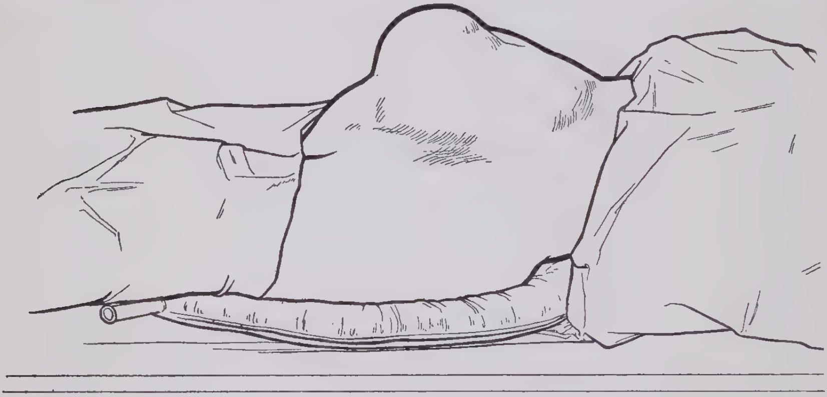


FIG. 5.—Sky line of abdomen in multinodular uterine fibromyoma. (After Kelly.)

The condition known as enteroptosis or Glenard's disease is easily distinguished, especially in the upright position. In all other conditions where swelling is present the abdomen projects abruptly forward, the hypogastric region being always above the upper line of the pubes; in enteroptosis it projects forward and downward, and the lower part of the hypogastrium falls below the line referred to. Moreover, there is in this condition, and in this alone, what is called a " bridle" line, a deep wrinkle in the abdominal wall, commencing above either iliac crest in the flanks, where it is but slight, but deepening, and becoming much more marked as it passes downward to its lowest level, about one inch above the pubes in the center.

The consistence of various tumors is very suggestive. The hard, nodular sensation conveyed to the examiner's hand in carcinoma of the pylorus; the firm, consistent, resistant, but not hard feel of a fibromyoma; the firm, semi-elastic resistance which suggests the presence of a solid mass enclosed in a more fluid medium of pregnancy; the entire elasticity of an ovarian cyst, are all well-marked variations which to a trained hand become almost diagnostic alone.

Many other points might be mentioned, but these are the chief; and as this lecture is already too long, they may be referred to in the next lecture, when the bimanual method of examination will be considered.

THE SURGICAL RELIEF OF TRAUMATIC EPILEPSY

A CLINICAL LESSON IN THE INSTITUTE FOR CLINICAL SURGERY OF THE UNIVERSITY OF ROME

BY D. B. RONCALI, M.D.

Assistant Professor of Special Surgical Pathology and Assistant to the Chair of Pathology in the University of Rome

Gentlemen: You all know very well how much I insist on the advisability of immediate trephining after traumatic lesions of the cranium whenever a depression of the skull exists, whether this is accompanied by a solution of continuity of the soft tissues or not. My teaching is that even though the compression of the brain may not at the time produce symptoms, it is certain sooner or later to have a hurtful effect upon the delicate nervous substance. In cases of depressed fracture, we should not wait for localizing symptoms to manifest themselves. Secondary trephining often either cures only for the time being, or sometimes fails to afford any relief. This failure seems to depend partly on the fact that the nervous elements have contracted such a habit of morbid explosive activity that the convulsions continue to recur in spite of the removal of the cause which originally produced them, and partly because the degenerative processes set up in the cerebral cortex as the result of compression become chronic, and complete return to the original physiologic condition therefore cannot be looked for with any confidence.

I have occasion, each year in this course, to state as my definite opinion that the therapeutic results of immediate trephining are as satisfactory as any operation in the whole range of surgery. The results of the secondary operation, especially when done at a much later period, are often incomplete, at times quite unsatisfactory, and sometimes, it must be confessed, instead of producing amelioration, the operation is followed by deterioration of the patient's condition and aggravation of his symptoms. The dangers of the immediate operation are less than those of subsequent trephining, because at the primary operation if infection should take place it will, as a rule, be extradural. In the secondary operation, however, there is a distinct risk of intrameningeal or intracephalic infection.

This teaching, then, makes clear the necessity for confidence in operating upon injuries of the skull complicated by depression of the cranial bones. On the other hand, I would not have you think that subsequent operations are entirely hopeless. On the contrary, not infrequently the symptoms of traumatic or so-called Jacksonian epilepsy are entirely relieved by the removal of a portion of depressed bone, or by the excision of a scar that is pressing upon the cerebral tissues, or by the levelling of an exostosis the presence of which has proved a constant source of irritation to the sensitive underlying cerebral tissues—perhaps for many years. At times, as you will see, even more serious pathologic conditions are relieved by operation.

I have occasion to show you this morning a patient who exhibited characteristic signs of traumatic or Jacksonian epilepsy, in whom operation was followed by complete relief of symptoms with absence of epileptic attacks for many years. Inasmuch as the patient presented a very characteristic history of traumatic epilepsy, I propose to consider the subject in order to impress upon you the lesson of how much can be accomplished, even after long delay, pointing out at the same time that there is assurance of relief when the operation is done at the time of the accident rather than subsequently.

Our patient is a young man, now 32 years of age, who was operated on more than 8 years ago. His history at the time he entered the clinic I shall read in detail, because it fully exemplifies this class of cases. About a year and a half before he came under treatment, as the result of a quarrel in a drinking place, he was struck on the head with a heavy stick. The blow affected particularly the left parietal region. He did not lose consciousness, nor was he deprived of power to walk. He was able to get to his house and went to bed, and says that he slept naturally. A few hours later, when he woke, he found that he was completely paralyzed in the right half of the body. Sensation and motion were entirely abolished. He suffered from aphasia and also from some deafness of the right ear.

He was carried to a hospital, where, however, he was treated without recourse to trephining. Six days after he received the injury he had his first convulsive attack. It occurred during the night, and he is not able to tell us much about it, except that it recurred three or four times before morning. Notwithstanding the daily recurrence of peculiar feelings resembling an aura, he began to improve in sensation and motility and gradually regained the use of the affected limbs. His deafness disappeared in about two weeks and his motion and sensation in the leg gradually grew better, the arm following in a short time. Later he once more acquired control over his facial muscles.

During this time he did not have any further attacks. After about 3 weeks he had improved so much that he was discharged from the hospital. He rested at home convalescing for 2 months, and then he once more took up his ordinary occupation—that of a country farm laborer. About 6 months after the accident, during a hot day in the middle of summer, while standing shortly after noon in the shadow of a tree to rest, he was taken with a new attack of epilepsy.

He says that from early morning he had not felt quite himself, being rather nervous and exhausted. The attack came on, however, without special warning. What he noticed first was a pulling at his nasolabial fold on the right side. This contraction was somewhat painful, and he could feel it spreading to other parts on the right side of the face and finally to the arm. Then he lost consciousness. The attack lasted only a few minutes, but was repeated several times at very short intervals, each new attack being at least as severe and as enduring as the first one. He was taken to a hospital and had no attacks for 8 days. He was given bromid of potassium, warm foot-baths, and ice to the head. He remained in the hospital for some 2 months. A few days after his discharge he had another attack, as severe as any, and this was repeated a number of times within a few days. He remained at home in bed for 3 or 4 days, with an ice-bag on his head, and then took up his ordinary occupation. From this time on he had attacks at irregular intervals of from 15 to 30 days, very rarely a single attack occurring, but usually two or three happening on the same day and always with the distinctive characteristic of being limited to the right side. The use of bromid of potassium proved of great service. The patient feels sure that it had the power in his case of prolonging the intervals between attacks.

The patient says that he had no means of telling beforehand when an attack was coming. Only a few moments before the actual attack, there was the warning aura which always consisted of contraction of the nasolabial fold. After an attack was over the patient remained for some hours in a lethargic state, not quite unconscious, but in a condition half awake and half asleep. For a day or two after he had an attack, coughing was followed by pain at the top of the head. Sometimes without having an actual epileptic attack, there was a certain confusion of mind during which he was apt to talk in a loud voice, to pull his ears with his fingers, and at the same time to exhibit certain rhythmic movements of his head from side to side. This condition lasted altogether not more than five minutes and was not accompanied by loss of consciousness nor by a true convulsive seizure.

I may say at once that the patient has no hereditary tendencies to any nervous disturbances either in the direct or in the collateral line. Before his first attack of epilepsy he never had any nervous affection, nor, for that matter, has he ever suffered from severe illness. Like most Italian country people, he had an attack of tertian malarial fever when he was a child, but this lasted only a couple of weeks and yielded completely to quinin. He had some enlarged glands in his neck, but these have been reabsorbed, and he denies entirely all venereal history.

When he entered the hospital the following notes were made with regard to his actual condition as far as it could be ascertained by objective signs: His cranium is orthocephalic with a slight tendency to brachycephaly. In its external confirmation it does not show any distinctive deformity. There is, however, a slight depression to be noted in the frontoparietal region on the left side. His frontal and parietal bosses are normally developed. In the neighborhood of the depression, toward the temporal region, there is a slight elevation of the skin on which can be traced a linear cicatrix. This is about 10 centimeters long and only 1 millimeter or so in breadth; it is whitish in color with certain portions of pinkish hue. Over the cicatrix there is entire absence of hair. This scar occupies exactly the parietal region on the left and is just in front of D'Antona's biauricular line. Palpation over the parietal region on the left side shows that the skin is of normal consistency and easily movable in every direction. The cicatrix is distinctly different in feel from the neighboring tissues. Palpation of the bone beneath the cicatrix shows all along the line of the scar an elevation of the margins except at the higher portion of the cicatrix, where a distinct sulcus can be felt. On deep palpation, there is some pain over the lower part of the cicatrix, though ordinary pressure produces no complaint. Percussion does not cause special discomfort either over the cicatrix itself or over the rest of the skull in this region.

The general condition is excellent. The patient's muscles are very well developed and seem to be perfectly symmetrical on both sides. The contractility is equal on both sides of the body. He seems to be perfectly able to accomplish voluntary movements without any awkwardness, and allows all passive movements without discomfort. With his eyes closed he is able to put his hands in any position without difficulty or excitement, and without tremor. His pressure sense seems to be equally well preserved on both sides. The face of the patient does not seem to be entirely unaffected by his ailment. On the right side there is somewhat less expression, and there is a distinct lowering of the labial angle. Closure of the mouth, however, is accomplished perfectly.

The patient assures us that lachrymation gives some difficulty on the right side, tears being much more likely to run over the lids and down his cheek. He is much more likely to have saliva flow out of his mouth on the right side than on the left. When we ask the patient to contract his lips in a broad smile, or so as to show his teeth, it is rather easy to see that the muscles on the right side of the face do not act as well as those on the left. There is no deviation of the tongue, however, although there is evidently some involvement of the nerves leading to the cervicofacial muscles, for the man has had a tendency to turn his head to the other side since the development of his epileptic attacks.

It is not unusual in these cases, when a patient has been much examined and has heard a good deal said by nervous specialists about his condition, to have certain functional symptoms develop in connection with the organic nervous condition. There may be, particularly in women, a hysterical addition of symptoms. This is especially likely to occur in those of nervous heredity, of which there is no trace in this case. We do not find any disturbances of sensation. There are no anesthetic or paresthetic patches. Tactile sensibility is equal on both sides of the body. Tests made with Weber's compass in order to decide acuity of sensation by detection of the distance of two points from each other, show no material difference in the sensibility of one side from the other. Pressure sensations are easily perceived, and the patient recognizes even light weights on the right quite as well as on the left side. Pain sense also is perfectly normal. Pin pricks are felt with equal intensity on both sides of the body, according to the amount of pressure employed. His thermic and frigorific senses are perfectly preserved, as can be seen by his immediate recognition of warm and cold test tubes. His special senses, except his hearing, are undisturbed. His smell is normal and his tastes for various substances are not in any way interfered with, nor do they differ on the two sides of the tongue. An ophthalmoscopic examination shows that there is nothing pathologic to be detected in his eye-grounds.

Notwithstanding the fact, then, that there is serious disturbance of his nervous system, most of the functions of his higher centers are perfectly preserved. His lower reflexes, however, are not so normal. The cutaneous reflexes are present but differ somewhat. His cremasteric reflex is quite lively on the left, but is lacking on the right side. His abdominal reflex is lively on both sides, though a little bit more so on the left. His periosteal reflexes are not accentuated either on the right or the left. His patellar reflex on the left is perhaps a little more lively than normal. There is a distinct exaggeration of it on the right. Ankle clonus exists somewhat on both sides, but is quite well marked on the right. You see that as we go farther from the brain there is more evidence of a certain lack of proper regulation of nervous reflexes. When we carefully make the various measurements of the skull, using D'Antona's method, we find that the farthest anterior portion of the cicatrix in the scalp corresponds almost exactly with the foot of the third frontal convolution. This would bring it almost exactly over the speech center of Broca. The extreme posterior end of the cicatrix is situated slightly beyond the Rolandic fissure, about at the point where its median third joins its lower third. The cicatrix, then, in its general position is almost exactly over the ascending frontal convolution in its lower third.

It seemed advisable then to attempt the relief of this patient's symptoms by an operative procedure. The circumstances that brought us to that idea were mainly his youth (he was scarcely more than 20), the fact that as time went on his attacks were becoming more frequent, that bromids seemed to be losing their power to control them, and that his general condition of mind and body were being disturbed by the recurrence of the attacks. We found great encouragement for operation in the generally good physical condition of the patient. He was of strongly built skeleton and muscular system, with good panniculus adiposus and perfectly healthy color. His lungs and heart were in good condition and he had never suffered from any symptoms from his digestive tract. An operation, then, would probably be borne well and be recovered from promptly and completely.

Accordingly the patient was allowed to remain under observation in the wards of the clinic for some weeks, in order that we might see his symptoms and judge of the severity of his attacks. Needless to say, this is an important preliminary to any operation of this kind, for, while the history may sometimes seem to indicate the necessity for an operation, the surgeon will find that the attacks are so different from what they have been described that he may judge an operation inadvisable or his ideas of the localization of the lesion may be modified.

During the time passed in the clinic the patient had two epileptic attacks, one the second day after his entrance, the other 10 days later. It was not difficult to notice that the aura, which was represented by a drawing of the nasolabial fold on the right side, was a real and not an imaginary movement. For a few minutes before an attack it gave the patient a queer sardonic look on this side of the face. The aura usually lasted for 3 to 4 minutes. During its existence the patient did not lose consciousness, and although he lost control of himself somewhat he was able to warn those standing near him that he might fall. At the time that the aura occurred there was a disturbance of the circulation and consequently of the color of the face. The patient became very pale. This pallor of itself was quite different from ordinary lack of color in the face. Usually the extremities were rather cold at this time. As soon as the contraction of the nasolabial folds became marked the epileptic attack itself, with complete unconsciousness, came on. The patient fell at full length, usually with the head turned somewhat toward the left side.

As soon as the contractions began to affect the arm there were involuntary movements of the trunk and of the hands. The hand was usually firmly clenched at the beginning and then there were some clonic movements of the fingers, followed by firm clenching once more. Then movements occurred in the lower part of the arm. After this the arm was raised and usually adducted and the fingers began to be worked in an athetoid manner. It was only after the clonic movements of the arm had proceeded this far that the right leg began to be contracted, and there was then some working of the toes. The clonic contractions in the leg were, however, much less marked than those in the arm. The arm and leg muscles, moreover, were not the only ones affected, for if the patient was examined naked it could be seen that there were alternate contractions and relaxations of the muscles of the abdomen and of the thorax. The contraction of the thoracic muscles was enough to hamper breathing, so that the countenance became darkly suffused. The cyanosis of the lips was startlingly distinct and had often been a source of worry to the patient's friends. A large quantity of frothy whitish sputum came from his mouth. The eyes remained half open during an attack, the pupils mydriatic to a slight degree, and the cornea perfectly insensible.

How complete the attack was may best be judged from the fact that a slight amount of opisthotonos with some lateral deviation toward the left was usually noticed. The right limbs contracted forcibly at times, but the contraction could be rather easily overcome. During this condition of right contraction the limbs on the left could be passively moved without presenting any resistance. The whole attack did not last much longer than a minute and a half to two minutes. The end always came with a series of stertorous respirations, during which a large quantity of saliva was thrown out of the mouth, coming almost as if it were sprayed with some force.

After this the color of the face became normal in a very few minutes. The patient did not, however, entirely recover consciousness at once. Even when the attack was over he remained in a sort of stupor, did not reply when spoken to, required considerable shaking to secure his attention, and remained in a sort of sleep waking state, which has been spoken of as *dormivigil*. After 4 or 5 minutes his intelligence returned, but a certain torpidity of intellect and a stupid condition remained for some time.

His subjective symptoms were characteristic of a true epileptic attack. He was very fatigued and utterly incapable of taking up any serious work or thought. He wanted to be left alone and his only wish was to sleep. This state of prostration usually lasted for an hour or two. The patient confessed, however, that he did not feel entirely himself during the rest of the day. It required at least one good night's sleep to restore him to his ordinary state of feelings, and if he had several attacks in succession, as was not unusual, the after-fatigue was increased, and he might sleep for many hours of a day and night before he was quite himself again.

The diagnosis in the case was perfectly clear. The patient was suffering from traumatic epilepsy affecting his right side and due to the irritation of the motor centers on the left side of the brain, because of the pressure upon them by the depressed portion of bone lying beneath the cicatrix that has been described as existing in his frontoparietal region. It was evident that, if he was to be relieved of traumatic epileptic attacks, this source of irritation must be removed.

Accordingly, after the patient had been under observation for about three weeks, the operation was performed by Prof. Durante. The incision in the scalp was made along a line corresponding to the fissure of Rolando as this is located by D'Antona's method. This incision was about 10 cm. in length. At right angles to it, at both ends, shorter incisions of about 3 cm. were made. The two flaps of scalp thus formed were raised and then with Macewen's osteotome two osteoperiosteal flaps were also raised. A portion of bone lying between the two bony flaps was removed for the moment. The thickness of the cranium as well as its consistency were found to be normal. Inspection of the dura mater showed that it was not only thickened but adherent all along the line of the old scar to the vitreous table of the skull. In the lower part of the old wound, where the bony fracture had been, the dura mater was absent over an area equal to the size of about a two-soldi piece (one of the old-fashioned two-cent pieces). At this point there was a cavity in the cerebral substance about the size of a mediumly large chestnut. This cavity contained some serosanguinolent fluid. There was in the fluid some portions of bone, one of them still adherent to the vitreous table at the lower end of the old fracture. Some smaller fragments of bone floated free in the little cavity. Around the cavity the brain substance was of not quite normal consistency, but was rather gelatinous. We could not determine exactly to which convolution the cavity corresponded, but it seemed to be in the lower third of both Rolandic convolutions.

The cavity was thoroughly washed out with boric acid solution and the surface of the brain and the neighborhood were also washed. All portions of bone in the cavity were removed and some detritus, perhaps from broken down soft tissues, were gently sponged away after the boric acid solution had been used. All the bleeding points were faithfully caught and tied, and when we were assured that no hemorrhage would take place the dura was brought together over the area. The osteoperiosteal flaps were then secured in place and afterward the scalp flaps were carefully sewed together.

That evening the patient had the aura that usually precedes an attack of his epilepsy; that is, there was a distinct contraction of the nasolabial fold on the right side, but nothing further than this. During the night after the operation the patient was rather restless and toward morning the breathing was inclined to be stertorous. Though they were carefully watched for, no real epileptic movements occurred with this. The aura was frequently repeated during the following day, but the epileptic convulsion did not develop. Toward evening the patient became more restful and the aura was noted no more. There were no nervous symptoms of any kind after the first 36 hours.

For several days after the operation there were occasional tendencies to contraction of the nasolabial fold, but this never went beyond this. The patient suffered from a slight attack of influenza, which was, during that year, 1891, so prevalent all over Europe and so frequent in all the hospital wards in Rome and especially among patients submitted to surgical operations, but this cleared up after a few days. On the twelfth day the dressings were removed—there had been no indication for their removal before, as the patient had suffered but slight discomfort from the wound, and that only during the first two days. The scalp incision was found to have healed by first intention. The dressings were renewed and removed some 5 days later and revealed the field of incision perfectly healed.

For some 3 weeks after the operation the patient remained in the clinic constantly under observation, but his condition was found to be always perfectly normal. It was then considered that he could be allowed to leave the hospital with entire propriety. Just before his dismission the site of operation was carefully examined and the bony walls were found completely restored. The consistency of the cranium at this point seemed to be quite equal to that of neighboring portions of the skull. Firm pressure made over the operative incision did not produce any sense of discomfort nor did it give rise to any appreciable nervous phenomena.

I show you the patient this morning in order that you may see and hear for yourselves the condition in which he is more than 8 years after the operation. The cicatrix is visible, but only when the surrounding hair is pushed aside in order to uncover it. The patient has no difficulty ordinarily in concealing its presence. It is a simple linear wound and beneath it the skull can be felt perfectly smooth and without any evidence of the depression that for nearly 2 years existed there as the result of the original injury. Pressure on this part of the skull does not produce any special discomfort, and percussion gives the same note and produces no more painful sensation than over the corresponding part of the skull on the opposite side of the head.

With regard to the nervous symptoms and his epileptic attacks, you will have to listen to his own story. He says that since the operation there have been no convulsions and he has had no unpleasant feelings at those points of his face in which before operation the aura of his epileptic attack always occurred. His general nervous condition is entirely restored. His patellar reflexes are both present and not exaggerated. Ankle clonus is absent on both sides. When I have him show his teeth there is no disturbance of the use of his muscles on either side of the face, nor that tendency to blankness of expression on the right which was noted before the operation.

Here, then, is a case which illustrates very well how much may be accomplished by operation even a long time after the original depressed fracture has taken place, and at a time when the nervous system, as the result of a series of epileptic attacks, might be expected to have acquired the habit of having these nervous explosions. As I said at the beginning of this morning's lesson, it is much better to avoid the risk of having these nervous symptoms develop by trephining as soon as a depressed fracture comes under observation. We must not feel, however, that the case is hopeless of operative relief because many months have passed since the occurrence of the fracture. In a word, for young patients particularly, the opportunity must be afforded them of getting rid of a serious nervous condition like this that may lead to general deterioration of even the higher nervous centers.

Pediatrics

CIRRHOSIS OF THE LIVER IN CHILDREN, WITH NOTES ON THREE CASES

BY W. F. HAMILTON, M.D.
Lecturer on Medicine, McGill University, Montreal; Assistant Physician to the Royal Victoria Hospital, Montreal, Canada

Case I.—C. S., female, aged four years, was first seen in the Out-Patient Department of the Royal Victoria Hospital, in May, 1897. Her mother brought her for treatment of cough and "cold," with which she had been ill for some ten days, her previous health having been good.

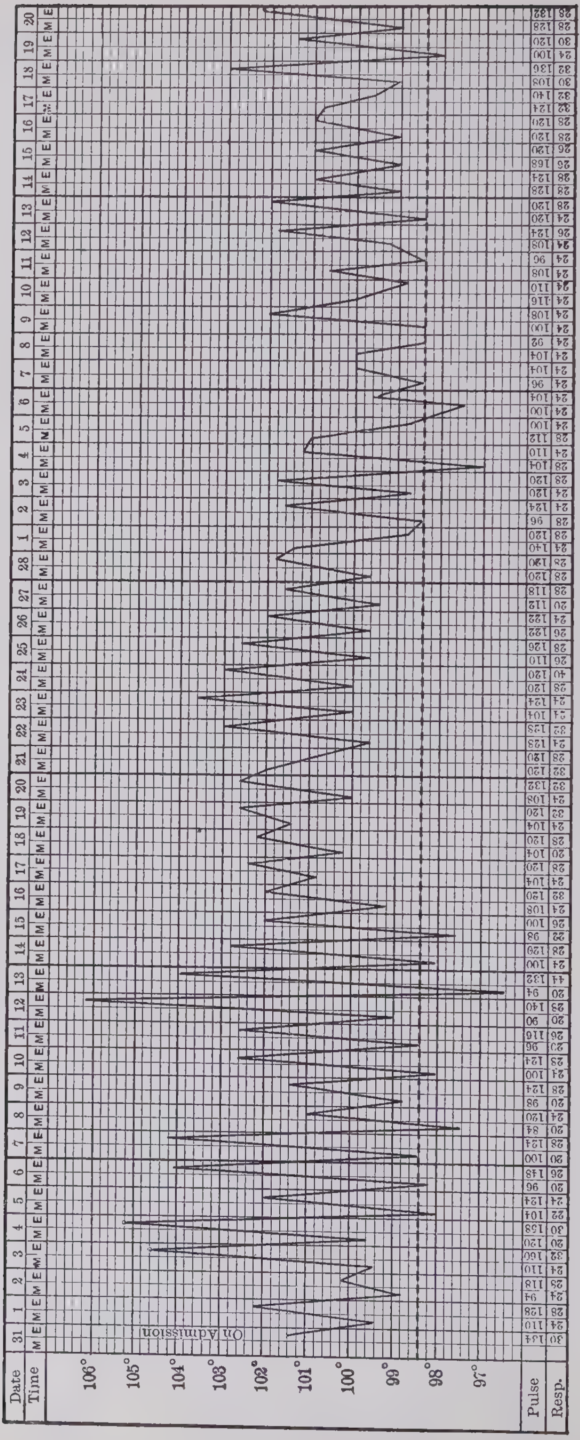
On examination at this time she was found to be well nourished, though somewhat pale, with icterus of the sclerotics and a yellow cast of the whole surface. The lungs showed a few signs of bronchitis.

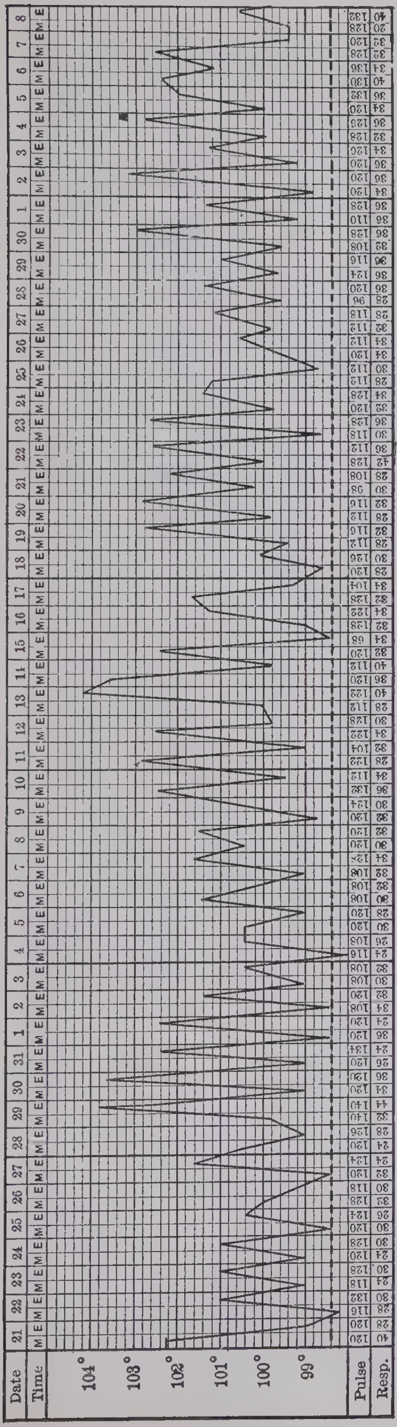
For several months after the first consultation the patient was apparently quite well. At the end of 20 months she returned with the complaints of headache, swelling of the abdomen, pain in the stomach, and cough. Frontal headache was often sufficiently severe to prevent sleep; abdominal pain, chiefly nocturnal, sometimes following the taking of food, at other times bearing no relation to this act, was generally referred to the umbilical region, and often was so severe as to induce wakefulness and provoke crying. Vomiting, diarrhea, and hematemesis were absent up to the time of the second visit, when she was admitted to the hospital.

The family and personal history of the patient afforded no suggestion of inheritance of syphilis or tuberculosis, nor of any illness likely to induce abdominal enlargement with jaundice. Her parents were living and well. She was born in Montreal, and had never been ill with any of the diseases common to childhood. There was no history of beer-drinking or wine-drinking.

ROYAL VICTORIA HOSPITAL, MONTREAL, CANADA

Caroline S., aged 4 years. Admitted January 31, 1899

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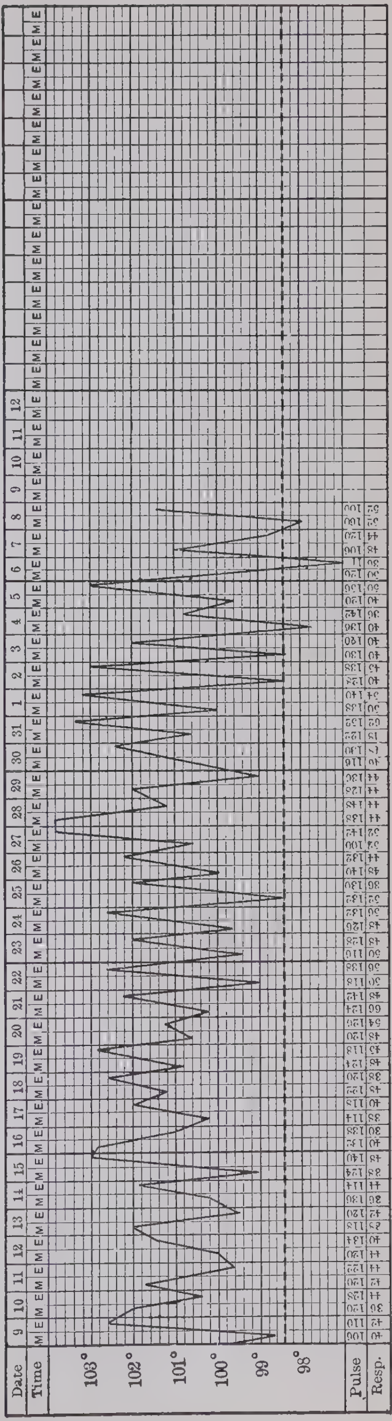
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On admission she was delicately formed, fairly well nourished, but rather pale and slightly icteroid. She was febrile (101° to 102° F.), the pulse rapid (110 to 134), the respirations practically normal (24 to 30). There was diarrhea (two to four stools daily), and there was cough, but no expectoration. The veins over the thorax were very plainly seen and somewhat enlarged; there was impaired resonance at the bases of both lungs posteriorly. At the apex of the heart the first sound was obscured by a murmur, transmitted toward the axilla; a murmur of the same rhythm was heard at the pulmonary cartilage.

The abdomen was prominent, chiefly in the upper quadrants; the superficial abdominal vessels were dilated. The circumference just above the umbilicus was 20¼ inches. The stools were clay-colored and of foul odor. On palpation of the abdomen in the hepatic region, 1¾ inches below the costal margin, the edge of the liver could be felt, sharp, not tender, and extending across the abdomen, to be lost beneath the left costal margin 1½ inches to the right of the middle line. One could readily distinguish the fissure between the right and left lobes. Some irregularity was palpable over the right lobe. Dulness of the right side began at the fifth rib. The spleen was enlarged, the notches distinctly palpable.

To follow this case minutely would consume too much time, so that we may review but the chief events which marked the progress of the disease. The patient was under observation from January 31 to June 8, 1899, a period of 129 days. She slowly lost in weight and strength and was constantly febrile; the range of temperature was variable (see Fig. 1), from 98° F. to 101° F., 102°, 103°, 104°, 105° F., and on one occasion it reached 106.2° F., to fall within the next 14 hours to 96.2°. Again the temperature curve would conform to that of continued fever from 100° to 102° F.; then a remittent type, 99° to 102° F.; then again an intermittent type, 98°, 101°, 103° F. In this connection it is important to observe that the paroxysms of abdominal pain and the great range of temperature were closely related, and when the pain was moderate the temperature curve would not run so high. The marked changes in temperature were not attended by chills or sweating.

Diarrhea was a constant feature of the case. The stools were sometimes completely acholic, at other times irregularly stained, partly white, partly yellow; and again completely stained,—uniformly yellow. We sought to establish, by observing the stools, the signs favorable to the theory of biliary obstruction, and at one time it was thought that the recurrence of signs of biliary matter in the stools after a period of acholia, simultaneously with the cessation of paroxysmal pain and high ranges of temperature, favored this view (February 14 to March 19). The stools during March were occasionally streaked with blood, and slight epistaxis occurred at intervals throughout her stay in the hospital. Vomiting was not frequent.

An increase in the circumference of the abdomen was observed, and on the 18th of April evidences of fluid in the peritoneum were undoubted. Bile was not constant in the urine. The cough persisted; the expectoration, always scanty, was repeatedly examined for tubercle bacilli, but yielded the same negative results throughout. Dulness over the left lung persisted, and moist râles were heard at the bases on both sides. There was no fluid in the left thorax on June 2. On May 30 the jaundice was noticed to become much deeper. The patient was discharged at the request of her parents on June 9, 1899.

During the next few days she became much weaker, while the abdomen became more distended. In association with Dr. J. J. Ross, who took charge of the patient on her leaving the hospital, aspiration of the peritoneal fluid was performed, and on examining the abdomen immediately afterward the liver was not palpable below or beneath the right costal margin. We were much surprised with this finding, for but a few months before this organ came about two inches below the costal margin.

On the 14th day of June the patient lapsed into a stuporous condition from which she did not recover, death supervening on the following day. A partial autopsy was made on the evening of the 15th, permission being obtained for but an abdominal incision.

A small quantity of yellowish, somewhat turbid fluid was found in the peritoneal cavity. There were no adhesions nor signs of chronic peritonitis. The spleen was considerably enlarged. The liver was found beneath the ribs, its lower edge above the costal margin. It was very firm and much smaller than normal, measuring 4¾ x 4½ x 3 inches. The left lobe was represented by but a remnant of fibrous tissue; the surface of the right lobe was covered with nodules of various sizes (Fig. 2). The color was olive green. It cut with much resistance, and the greenish-yellow surfaces thus produced showed numerous invading lines of fibrous tissue enclosing areas of liver tissue of various sizes, and showed also that the left lobe was almost entirely composed of fibrous tissue (Fig. 3). An examination of several microscopic sections revealed an extensive cirrhosis, characterized by—

1. The formation of abundant connective tissue, irregularly arranged, and not so as to define a cirrhosis of the so-called unilobular type.
2. The formation of new bile ducts.
3. Distinct thickening of arterial walls, an endarteritis; and
4. Dilatation of the lobular capillaries, variable in degree, in different parts of the same lobule. The liver cells in some places showing signs of advanced atrophy.

The general appearance of the organ, with the evidence afforded by the microscope, warranted the classification of "mixed cirrhosis." The microscopic appearances are rather in favor of biliary or obstructive cirrhosis, but the gross appearances of the organ and the clinical history of deepening of the jaundice late in the disease, as well as the sharply defined edges of many of the lobules, are what one sees more frequently in ordinary portal cirrhosis.

The gall-bladder presented no evidence of cholecystitis. The larger bile ducts were patent.

Case II.—T. B., male, aged eight years, first came under medical care in the Royal Victoria Hospital on July 30, 1896, complaining of cough, vomiting, and swelling of the abdomen. His mother stated that her son's illness began early in January, 1896, with distressing dry cough, scanty expectoration, severe intermittent abdominal pain, night sweats, and vomiting after a fit of coughing or after meals. This did not last more than three or four weeks; and although the severity of the symptoms subsided, his appetite remained poor and he continued to get thinner. About the middle of May, increase in the abdomen was first noticed, and had been progressive until patient was admitted. Before entering the Royal Victoria Hospital he spent some fifty days at another institution in the city, where persistent cough and diarrhea were the chief clinical features.

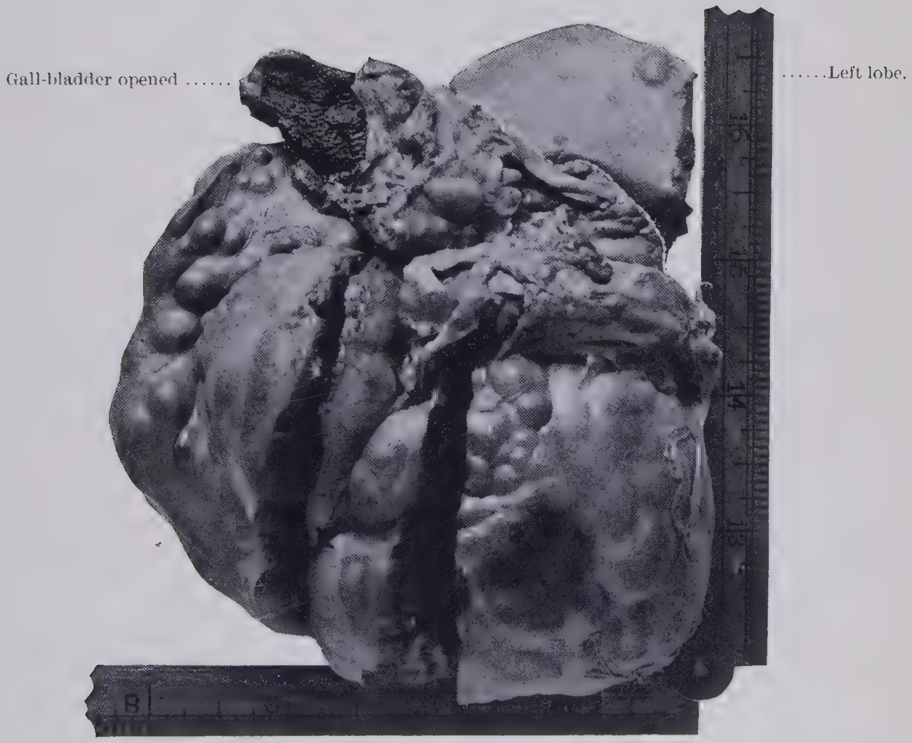

FIG. 2.—Illustrating the diminution in size, the irregularity, and the nodular appearance of the liver in Case I.

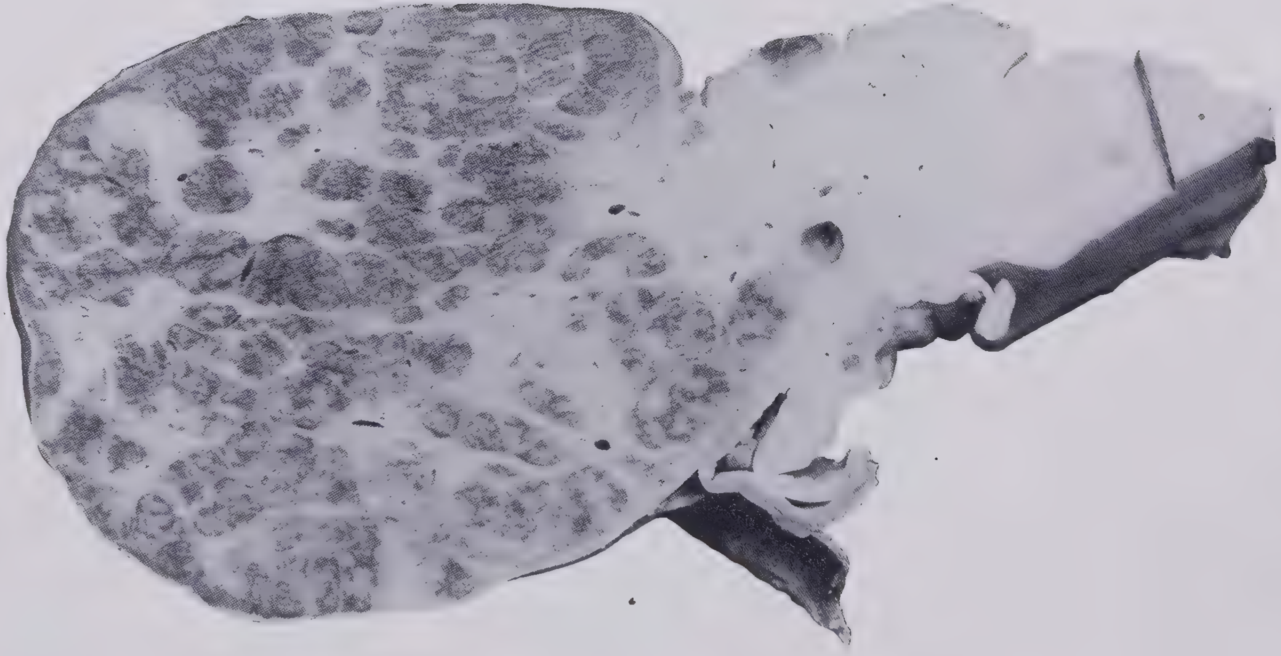

FIG. 3.—Longitudinal section of the liver of Case I.

The patient was born in England, coming to Montreal at the age of two years. When five years of age he had scarlatina. With this exception he was always healthy up to January, 1896. He had never used alcohol in any form. His family history was negative as to syphilis, tuberculosis, and carcinoma.

When he came under our observation he was a poorly nourished, well developed, and very intelligent boy. The skin and mucous membranes were rather pale, the abdomen prominent, and the legs edematous.

The examination of the circulatory system yielded no evidence of disease. The pulmonary second sound was accentuated; however, this was doubtless due, in part at least, to the presence of fluid within the chest. There were signs of fluid in both pleuras, dulness rising much higher on the left. The abdomen was prominent and tense. The superficial veins over the chest and abdomen were considerably dilated. Signs of ascites were present, the dulness varying with change of position. The liver was palpable on "dipping;" its surface felt smooth and there was no special tenderness; the lower border in the mammary line was about on a level with a horizontal line passing through the umbilicus.

The spleen was not palpable. There was considerable difficulty in breathing, for relief of which the patient almost always sat up in bed. On the 6th of August an attempt was made to relieve the dyspnea; the right pleura was aspirated and 36 ounces of clear, straw-colored fluid were removed. On the 14th the peritoneum was aspirated, and but 12 ounces of greenish, slightly turbid fluid were withdrawn. This opportunity of a more complete examination of the abdomen was seized upon; the lower border of the liver was palpable just below the umbilical line; the edges were rounded and the surface smooth, except one area over the right lobe.

First admitted on July 30, 1896, the patient was discharged from the ward on December 24, 1896. During this stay in the hospital the right pleura was aspirated twice, and 85 ounces of serous fluid were withdrawn. The abdomen was tapped three times, and about 100 ounces removed. The temperature curve rarely passed beyond 99° F., and was chiefly subnormal. A mild degree of diarrhea with frequent vomiting marked the intestinal and gastric disturbances. There was no icterus and bile was never found in the urine.

During the remainder of his life—18 months—the patient was admitted to Ward C five times, spending the most of his time in the hospital, in all about 275 days. The abdomen was tapped eight times during this period, making in all eleven tappings, by which upward of 38 pints of serous fluid were taken away.

The size of the liver became appreciably smaller; after the first tapping the lower edge of the right lobe was palpable somewhat below a horizontal line passing through the umbilicus. After the tenth operation this part was palpable about 2 cm. from the costal margin in the right nipple line; while from the ensiform cartilage to the hepatic margin 6 cm. covered the distance. The spleen was never palpable.

It was at first thought that tuberculosis was present in this patient. Frequent negative examinations of the sputum and the absence of reaction to the tuberculin test excluded this. Icterus was never marked in this case, although present for several months. Urobilin was detected by Huppert's test on two or three examinations.

The temperature curve, which occasionally rose above 99° F., became quite irregular during the last two weeks the patient was under observation, assuming a low intermittent type, the maximum height of which was 102° F. He died about July 1, 1898, just two and one-half years after the onset of his illness.

In this case no autopsy was obtained.

Case III.—A. K., aged seven years, female, first sought relief at the hospital on October 8, 1900, when she complained of swelling of the abdomen, weakness, and loss of appetite. Her mother stated that though never very strong, the patient had been in her usual health up to April, 1900, when she had an attack of measles. Thereafter she was never so well as before. In July an attack of vomiting and diarrhea had been followed by deep jaundice, which had now pretty well faded. After this attack her abdomen began to swell, and had gradually increased in size until the date of admission.

The child had always lived in Montreal. There was no alcoholic history whatever and her diet had been of the plainest. The family history was negative, with the possible exception that the mother had had three miscarriages.

From her appearance one would consider the patient beyond seven years of age. The skin was of a dark, dull, earthy color, with a faint yellowish tinge; the sclerotics were clear; the lips cyanotic, and the fingers clubbed.

The temperature was 99.4° F., pulse 120, and respirations 24. There was considerable emaciation, but no edema. The glands of the neck and of the axilla were somewhat enlarged. The lungs and heart gave no evidence of chronic disease. The mind was clear.

The appetite was gone; the teeth were sensitive, the gums bled easily; the tongue was moist and coated, but red at the tip and edges; the bowels were inclined to be constipated. The abdomen was greatly distended, measuring at the umbilicus 67.5 cm. and at the lower costal margin 71 cm. The skin over the anterior part was tense and shining with the superficial veins showing plainly through. The protruding umbilicus and bulging sides, fluctuation, and movable dulness indicated ascites. The liver was palpated with difficulty. The spleen was much enlarged, its upper margin at the seventh rib in the mid-axilla, its total length being 5½ inches. There was no tenderness on palpation.

The urine presented but a trace of albumin, but no pathologic pigment and no casts.

The blood showed, erythrocytes, 4,100,000; leukocytes, 18,000; hemoglobin, 70 per cent.

Admitted on October 8, 1900, the case terminated in death on the 17th of that month. During this time there was very slight bleeding from the tongue; the stool occasionally was streaked with blood. Vomiting supervened on the 15th, and the vomitus was blood-tinged. The temperature, which up to the 14th was between 98° and 99° F., ran to 104° F. on the 15th, and the patient became stuporous, with retraction of the head and irregular aimless movements of limbs and body.

On the afternoon of October 15 the abdomen was aspirated for the first time, and 40 ounces of yellow fluid withdrawn. The patient did not regain consciousness, dying early the following morning.

The post-mortem examination revealed among other conditions the following of most importance: The pleural cavities contained a small amount of slightly blood-stained serum. There were no adhesions nor sign of tuberculosis. The peritoneal cavity contained about 900 c.c. of dark straw-colored fluid. The liver was retracted behind the costal margin and showed a marked hob-nailed appearance. The stomach was large and congested. Between the upper surface of the liver and the diaphragm there was a considerable quantity of fluid. The vessels of the lower part of the esophagus were congested and very prominent in the last two inches, where, when held to the light, a plexus of dilated veins was recognized. The mucous membrane of the stomach was swollen, reddened, and generally congested, and at the cardiac end were small irregular submucous hemorrhages. In the upper part of the jejunum rare submucous hemorrhages were seen. The duodenum and jejunum were congested.

A more detailed examination of the liver is of interest. It was 17 x 11 x 5 cm., small and contracted. Both lobes were atrophied to about the same extent and upon the surface presented in a marked degree the hob-nailed characteristics (Fig. 4). Round, pale, yellowish nodules projected nearly everywhere. There were no adhesions, but the veins on the ligamentous continuation of the left lobe were prominent. A smooth thickening of Glisson's capsule was observed beneath the left lobe. The organ was very tough, relatively dry and bloodless, and on section showed great numbers of pale yellow nodules, one-eighth to one-fourth of an inch in diameter, which stood out prominently against a paler pink and scanty background, and projected above this. The portal vein was found relatively small, the bile ducts impervious, and the bile thick, viscid, of a greenish-black color.

The spleen was 15 x 9 x 5 cm., firm and congested.

Microscopic sections of the liver were characteristic of Laennec's atrophic multilobular cirrhosis, with abundant formation of fibrous tissue.

Remarks.—The diagnosis in Cases I and III was amply confirmed, as has been already shown, by a description of the specimens and reference to the photographs herewith reproduced. The diagnosis of hepatic cirrhosis in Case II may admit of discussion. When first seen the case-picture suggested several possible conditions as explaining the patient's complaints. Among these may be mentioned tuberculosis, tuberculous peritonitis, perihepatitis, pseudo-cirrhosis, and true hepatic cirrhosis. It will be remembered that in this case we had a patient, aged eight years, who had been

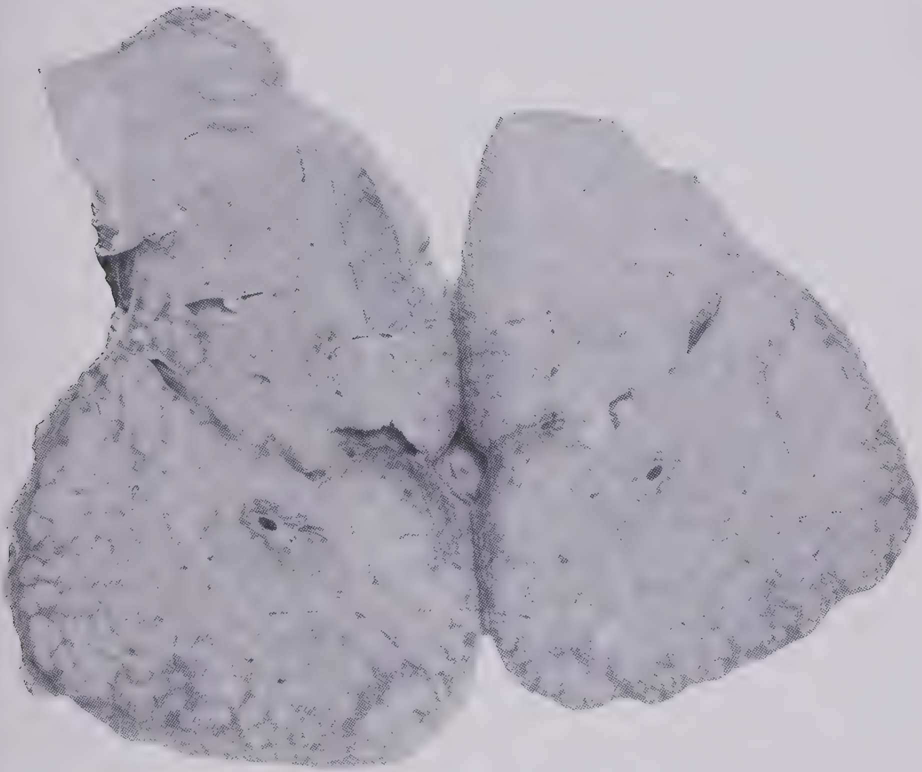

FIG. 4.—Longitudinal section of the liver, from Case III.

ill for upward of nine months with a disease, acute in its onset, marked by a febrile temperature, cough, vomiting, slight expectoration, night sweating, and abdominal pain, followed by dyspnea, and, later, enlargement of the abdomen. This passed into a chronic state, marked by ascites, hydrothorax, edema of the legs, and emaciation.

The family history lent no support to the view of tuberculosis, and the negative result to the tuberculin test set it aside. Perihepatitis found but few if any points in its favor and so many points against it—as, for instance, the hepatic enlargement and the patient's age—that it was dismissed. Pseudo-cirrhosis (of Pick) could not be established, and by exclusion a diagnosis of hepatic cirrhosis was decided upon. The findings at that time were not against this view, while the course of the case as described above affords it ample confirmation.

Before discussing the etiology, etc., of cirrhosis of the liver in children, I desire to point out that Cases I and II, which we were able to follow from the earliest to the latest scenes, illustrate the early enlargement and the late shrinkage of the liver. In the first case the hepatic margin was palpable 5 cm. or so below the edge of the ribs in the right mid-clavicular line, receded to the costal margin and beneath it. This change was accompanied by the appearance of ascites. In the second case the enlargement was more pronounced, the liver remained palpable throughout the course of the disease, but yet the edge receded considerably from the position in which it was first described.

Another feature was the late development of ascites, and for the relief of this tapping was resorted to but *once*. At the partial autopsy made no peritonitis was found, and evidently the peritoneal fluid was purely hepatic. This observation supports that made by Hale White in his article on "Perihepatitis," in Allbutt's "System of Medicine:" "In cirrhosis the patient rarely lives long enough after the first tapping for a second to be necessary. The supervention of ascites in cirrhosis heralds death."

When we consider our second case with respect to this teaching, it may be that an explanation is found for the frequent tappings, and perihepatitis probably did exist, yet the case did not present the characteristic features of that disease alone. From the specific gravity of the fluid (1015, 1017, 1016, 1018, 1028, 1024, 1015) and the frequent tappings we have little more than scant evidence for the view that chronic peritonitis was present.

Cyanosis and clubbing of the fingers, probably most frequently associated with congenital cardiac disease, have been observed in cases of cirrhosis in children. Dr. H. R. Smith calls attention to this fact, finding corresponding signs in one of his cases, and referring to three such cases reported by Gilbert and Fournier, and one by Dr. Taylor.

Etiology.—On going into the history of these cases to establish the etiology, one found no evidence of any cause among the accepted causes of cirrhosis, such as alcoholism, syphilis, and rickets. The history in Case I is negative on all etiologic points. Case II had scarlet fever about two years before he came up for treatment, but it was impossible to ascribe to this illness the onset of symptoms of the hepatic disease. We may admit, however, that such a relationship may have existed. In the history of Case III a very close relation both in point of time and failing health exists between an attack of measles and the supervention of signs pointing to hepatic disease.

The theory that cirrhosis of the liver is due to infectious disease, though advanced years ago, is acquiring an increasing amount of evidence in its favor. Since 1877, when Klein published eight cases due to scarlet fever, several cases of cirrhosis in children have been reported showing a fairly definite etiologic relation to infectious diseases.

Case V, in H. R. Smith's series, had scarlet fever at eleven years of age, and never enjoyed good health thereafter, dying of cirrhosis of the liver at the age of 23 years. In 1890, Dr. Edwards, in the *Archives of Pediatrics,* records his belief in the relation of cirrhosis to measles and scarlet fever, remarking that this disease may be seen in all stages, and that it is possible for the cirrhotic process to become established or to disappear. In looking over the literature of other countries it appears that Laure and Honorat held this view concerning the origin of hepatic cirrhosis in certain cases. Henoch supported this view touching the etiology, when he reported the case of a child aged eight years who never recovered completely from an attack of measles, but suffered immediately thereafter from some digestive disturbance, within a week became jaundiced, showed an enlarged liver, the surface of which at the end of six months was rough and uneven. This observer, in considering the frequency of infectious diseases and this variety of hepatic cirrhosis, remarked that a restoration or recovery from the primary diseased condition as found in hepatic cells must occur.

The late Dr. Pepper gave additional support to this view in a report upon a case of hepatic cirrhosis in a child of eight years following an attack of measles, during which hepatic disorder was manifest in jaundice. Within a year the patient died, and at autopsy a typical hob-nailed liver was found. Dr. Crook, in speaking upon a case of Dr. Goodhart's reported before the Pathological Society of London in 1889, said that he had several times found extensive acute interstitial hepatitis in young children who had died from scarlatina.

With these observations upon the relation of infectious disease to hepatic cirrhosis it is justifiable to include measles and scarlet fever among the etiologic factors.

In an analysis of 63 cases of cirrhosis of the liver in children, the late Dr. R. P. Howard found that alcohol was active as a cause in 15.8 per cent., syphilis in 11 per cent., and tuberculosis in 11 per cent. Granting that 20 per cent. of all cases are due to the infectious fevers, there yet remains a large percentage unaccounted for—little less than half. Direct bacterial infection or the action of toxins may cover the balance of the cases, and to this view the recent work of Adami, Boinet, Ramond, and others, give strong support.

Classification.—If the attempt to arrive at a satisfactory conclusion concerning the etiology of these cases is difficult, a task even more difficult confronts one who would classify them clinically. In the first case such a variety of clinical features have been observed and the pathologic findings are such that it must be considered a mixed case. In this and similar instances may we not have to deal with transitional forms as suggested by Alexander James, which are most common in young subjects? Or may not a secondary etiologic factor favoring the development of cirrhosis of another type, as Senator suggests, give us one of the mixed forms puzzling the clinician and the pathologist alike? With Dr. Cheadle all will agree that " in our conceptions of disease we look for a strict separation into classes, and the expectation of close conformity to given types is apt to warp our intelligence."

The first case presented many clinical features bringing it under that class known as Hanot's cirrhosis. Indeed, for many weeks there were all the elements making up this case-picture—the enlarged liver; the enlarged spleen; icterus; pain, with intense exacerbations; irregular, sometimes high, febrile temperature; the absence of alcoholic history; and the absence of signs of portal obstruction. Then the picture shifted to that of cirrhosis with atrophy and signs of portal obstruction—deepening jaundice, toxemia, and death. The anatomic conditions have already been described.

Clinically, Case II was that of portal cirrhosis with atrophy following upon an early stage of hypertrophy, while the third case, both clinically and anatomically, was one of atrophic (Laennec's) cirrhosis. These three cases may belong etiologically to the infectious cirrhoses, and anatomically the first shows a mixed form, while the third shows the multilobular or atrophic type.

The annexed table may aid in a review of several of the more important facts in connection with these three cases.

Conclusions.—1. Cirrhosis of the liver in children, while rare, is being recognized more and more.

2. Scarlet fever and measles are to be included among the causes of cirrhosis.

3. There is, at least in some cases, a stage of enlargement followed by marked atrophy.

4. A typical clinical picture of Hanot's hypertrophic cirrhosis may pass over into one of atrophic cirrhosis with portal obstruction.

I am greatly indebted to Dr. Adami for his kindness and help in the post-mortem and microscopic examinations, and to Dr. Patrick for the photographs.

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TABULAR STATEMENT OF THREE CASES OF CIRRHOSIS OF THE LIVER.

Case.	Sex.	Age.	Duration.	Liver.	Spleen.	Jaundice.	Ascites.	Urine.	Stools.	Infectious Diseases.	Autopsy.
I.	Female.	Four years.	Two years.	Enlarged, then small.	Enlarged.	Present.	Late event.	Bile not constantly present.	Irregular colored, frequently acholic.	Negative.	Small, hob-nail liver; enlarged spleen; no obstruction of bile.
II.	Male.	Eight years.	Two and one-half years.	Enlarged, then small.	Never palpable.	Never marked, although present for several months.	Chronic.	Negative.	Normal.	Scarlet fever at five years.	No autopsy.
III.	Female.	Seven years.	Four months.	Always small.	Enlarged.	Present, not marked.	Present.	No pathologic pigment.	Grayish yellow, gray.	Measles at six and one-half years.	Small hob-nailed liver; spleen enlarged.

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Obstetrics and Gynecology

THE CAUSATION AND TREATMENT OF STERILITY IN WOMEN

A CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC HOSPITAL AND SCHOOL FOR GRADUATES IN MEDICINE

BY J. RIDDLE GOFFE, PH.D., M.D.
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GENTLEMEN: One of the most interesting subjects in the whole domain of gynecology is sterility. Nothing can be more gratifying to a physician than to be able to respond to the wishes of a patient who is anxious to have children, but who finds that there is some obstacle to maternity. There are two principal causes of sterility. One of these we now know as infantile uterus; the other is a chronic inflammatory condition of the pelvic organs which has left either purulent material or adhesions that bind down the tubes and ovaries and make it impossible for the ovum to find its way properly into the uterus, or for the spermatozoon to reach the ovum under circumstances favorable for fertilization.

Infantile uterus is a condition that we have come to understand properly only of late years. It was formerly known very commonly as anteflexed uterus. The condition is really one of imperfect development of the uterus in consequence of which it remains in the condition that is normal in females before puberty. Lawson Tait said that, as the result of his long experience in the treatment of the diseases of women, there was no conclusion firmer in his mind than that of devout thankfulness that he belonged to the sterner sex. Up to puberty boy and girl develop on equal terms. After that time there is in the woman a distinct diversion of much more vital energy to the genital tract than in the male, and this continues until the menopause. Unless there is a proper provision of surplus vitality in the system to support this additional strain put upon it, symptomatic conditions requiring treatment are certain to arise.

One of the most fruitful sources of so-called " woman's troubles" is undoubtedly the infantile uterus—formerly thought a deformity. The reason for the mistake is not far to seek. In the infantile condition, that is, up to puberty, the uterus being functionless is sharply anteflexed on itself. This looks like a deformity and, by analogy with such conditions as club-foot, it would seem that it should be remedied by surgical procedures. The club-foot is cut and violently straightened and then put in splints to retain it in its new position. The deformed uterus with anteflexion used to be treated in the same way. Marion Sims devised an operation based on this principle. He split the posterior lip of the cervix up to the internal os and then made an incision anteriorly at the junction of the cervix and the body of the uterus, the usual place of flexion, and inserted a glass plug to hold the uterus straight until the raw surfaces had been covered by granulations. In spite of the limited success obtained with this operation, Marion Sims continued to use it during his life, and considered that it was founded on a proper therapeutic principle.

I was an interne at the Woman's Hospital here in New York for several years while these operations were being performed. During my service as resident I did not see one patient improved by the procedure, and all of them to my mind left the hospital in worse condition than on entrance. The main reason for this was the lack of asepsis with which the operations were done. You must remember after all that this was 20 years ago. In practically all the cases some infection took place, and the consequence was pelvic peritonitis. This was, as a rule, of low grade, but occasionally even pelvic abscesses developed. The inflammatory condition set up caused a binding down of the tubes and ovaries by adhesions, and after weeks of suffering the patients were sent out, as a rule, more hopeless in their sterility than before and with genital organs certainly more crippled than before. Personally I resolved that I would never perform the operation.

Courty, of Montpellier, a French gynecologist, some 20 years ago wrote a book on diseases of women in which he brought out the fact that the so-called anteflexed uterus was really an undeveloped or infantile uterus. He had made many autopsies on fetuses and on young girls and found that anteflexion was constantly present before puberty. Hence he called this condition the infantile uterus. Subsequent investigations have confirmed this idea. We know now that unless proper development of the uterus occurs at puberty the opportunity for its evolution to the normal size is apt to be missed forever. As a result the uterus fails entirely to assume its proper shape and size, unless some unusual stimulus occurs to reawaken the slumbering tissues.

The symptoms produced by infantile uterus may be grouped under the two headings, dysmenorrhea and sterility. There is pain just before and during menstruation. This pain is not only in the back, but it is sometimes also referred to internal organs. Menstruation is likely to be scanty; it is usually irregular and sometimes absent, although there are menstrual molimina that give quite as much discomfort as if there was an actual menstrual flow. Not infrequently patients are so prostrated at the time of their menses that they have to take to their beds for several days, and this is apt to be accompanied by reflex symptoms, such as headache and flushing of the face, and nervous disturbances of various kinds, such as indigestion, skin affections that remain obstinate to treatment, and reflex interference with the general health.

It must be remembered that while we speak of infantile uterus, it is not the uterus alone that is undeveloped. In these cases the ovaries and tubes have also failed to reach their proper development, and this fact, which has often been demonstrated, would of itself be sufficient to make it clear that no cutting or splinting of the uterus will suffice to relieve the condition present or the many symptoms that are usually associated with it. What is done with curative purpose must affect the whole genital tract if it is to meet the indications present and prove really helpful for the pathologic condition.

If marriage takes place sterility not infrequently follows. It is true that occasionally impregnation takes place even through a more or less infantile uterus. When this does happen, there is usually an extreme degree of nausea and vomiting during pregnancy. In fact, the presence of an undeveloped uterus is one of the most frequent causes, I may say the most frequent cause, of the excessive nausea and vomiting of pregnancy. If gestation goes on to normal term in these cases, there is likely to be a laceration of the cervix when birth takes place. After all it is not difficult to understand why this should occur. The uterine muscle was originally in no condition to take on the exaggerated development required of it in order to accommodate a full-term fetus, nor had the cervix developed that elastic quality which enables it to dilate sufficiently to give passage to the child.

As we now know the cause of the condition and the symptoms that follow it, our treatment can be directed much more rationally than before. The most important part of the treatment is the prevention or prophylaxis of the persistence of the infantile condition of the uterus. This consists in taking proper care of young girls at and before puberty. The condition of infantile uterus occurs especially in girls who are intellectually ambitious and apply themselves very strenuously at school, or who are pushed very much in their studies. The brain develops at the expense of the general system. This is now well recognized by all physicians, and when the conviction of the relation of cause and effect between the two conditions of precocious mental work and failure of sexual evolution has spread also to mothers, much will be accomplished for its prevention.

Another class of individuals in whom infantile uterus is very common are young girls who have had to work too hard. Shop girls, for instance, who have to stand on their feet for long hours and who begin their work in stores as cash girls at 9 or 10 years of age and continue during the precious years of puberty, are especially likely to have undeveloped uteri. In the lower part of New York city there are night clinics for patients who cannot come for treatment during the day, either because they cannot afford to lose their wages or because they would be likely to lose their positions. In these dispensary services there is a large proportion of young girl patients suffering from the symptoms of infantile uterus, the cause of whose ailments may be found in the conditions of their employment.

It is easy to understand how the pathologic condition has developed. The girls become overtired, yet must go on with their work; the strain on them is too much for their nervous system, and then while on their way home in the evenings they frequently have to stand in the cars when already exhausted with the work of the day. Besides this they are poor and are poorly nourished. Then, too, they are foolish in the food they select. They are apt to be capricious eaters and to take things that are not good for digestion.

As a consequence, when puberty comes they have not the requisite surplus vitality to supply the energy for the extra development that must take place in the genital system. The result is the train of symptoms that we have described, because of the persistence of the undeveloped anteflexed uterus of childhood.

It is extremely important for the next generation that the health of young girls should be very carefully looked to as they approach the period of puberty. The physician can do nothing better for the next generation than impress this fact on mothers. The shop girls present an extremely difficult problem in this matter, but physicians should encourage, by every means in their power, the introduction of such amelioration of the life of the shop girl as may make her condition a little less unpromising. There is at the present time in New York an organization of women for the purpose of securing better hours and easier methods of labor for shop girls. This is so powerful that it has compelled the heads of department stores to furnish seats for shop girls, to be used whenever there is an opportunity to sit down. This organization has also begun a crusade for shorter hours, and has already accomplished something in this matter. Needless to say, doctors should encourage movements of this kind. Even with all the weight of social and professional influence there will still remain many abuses practically impossible of correction.

I am a believer in a great deal of outdoor life for girls at this important period of their life. Walking, riding, driving, are beneficial exercises, particularly because they take people into the air. In the formal walk, or ride, or drive, however, I am not much of a believer. There is very little value in exercise, even in the open air, unless it is accompanied by a helpful relaxation of mind and new mental interests that bring forgetfulness of care and stimulation of all the lower nervous centers. I believe, then, that outdoor games for girls are important, especially games in which the competitive spirit is aroused and in which as a consequence there is a complete forgetfulness of self in the healthful exercise that brings healthy tiredness.

I am not much of a believer in set gymnastic exercises unless they are accompanied by the pleasure of competition and there is something of the gaming spirit in them. One reason why gymnastic exercises should not, of themselves, be considered of too much value is that they are not, as a rule, performed in the open air, and as a consequence the most important element in all healthful exercise is lacking. The performance of a set series of gymnastic exercises may prove a mere waste of muscular energy without anything to stimulate the circulation or increase metabolism. Every precaution must be taken, then, to make gymnastics interesting as well as merely a means of muscle development.

Almost needless to say, physicians should encourage the organization of women's athletic clubs, so as to secure athletic opportunities for growing girls and to create the proper feeling with regard to the necessity for exercise. Fortunately at the present time there is a social element in the matter that has very much encouraged the formation of women's athletic organizations. For instance, in New York there are many women who do not consider that they or their daughters are taking proper care of their health unless they spend several hours a week in the gymnasium. This is already a very good beginning, and recent social interest in golf and in other outdoor sports, as well as the popularity of certain indoor sports, such as basket-ball and even the much joked about ping-pong, have their good sides.

The treatment of infantile uterus is not very definite and direct, but is undoubtedly helpful, and succeeds almost without exception in developing the genital tract nearly to the condition which it would have reached had there been a normal evolution of the genital organs at the time of puberty. Of course, this development is not accomplished instantaneously, nor in a short time. In the ordinary course of nature the uterine development would take several years. Even after puberty, then, to bring the uterus up to the normal requires a course of long stimulation. For this there is no specific treatment, but a series of therapeutic measures that fulfil the indications of increasing the blood supply to the part and consequently of stimulating trophic conditions and growth.

When patients come with an infantile uterus, I assure them that they can be cured, but that it will take at least 3 months of treatments given at least twice a week. The treatment is begun by gradual dilatation of the uterus with one of the ordinary dilating instruments, followed by the application of pure carbolic acid to the interior of the uterus. This is followed by the use of a glycerin tampon in the vagina, which is removed after 36 hours and followed by a hot douche taken once a day. The water for the douche must be as hot as can be borne, and should be in large quantity. To use a small amount of water will do more harm than good.

In infantile uterus there is usually found a sharp anterior flexion which has led to the retention of some, at least, of the menstrual secretion. As these blood-clots, blood serum, and exfoliated membrane form an especially favorable culture medium for microorganisms, it is not surprising that they should become infected. The result is the development of a chronic or subacute endometritis. Hence the necessity for thorough dilatation of the cervical canal which provides for effective drainage. The stretching of the muscular fibers in the wall of the cervix also softens the muscle in these parts, the irritation set up causes a freer flow of blood to the parts and stimulates the development of the as yet unevoluted muscle fiber.

All this may be done in the office and without the administration of an anesthetic. If patients are very nervous they will not be able to stand the pain, but as a rule there is not much complaint. At any sitting the dilatation should only be carried as far as can be borne without the infliction of pain sufficient to cause much complaint. The dilatation must be done very slowly. Aside from the thorough aseptic precautions, this is the most important element in the technic of the procedure. Rapid dilatation inflicts unnecessary pain and causes lacerations which increase the already excessive connective tissue and leave the patient more liable to septic complications. Needless to say, the manipulations should be done under the most scrupulous aseptic precautions.

Care must be exercised with regard to delicate patients, even when they are reasonably insensitive to pain. The reflex nervous effect of the insertion of the dilator may cause a patient to faint on the table. If a patient faints easily (and this should always be asked about), it is especially important to secure some local anesthesia before attempting dilatation. The local anesthetic employed may be either cocain or pure carbolic acid. Either of these may be applied to the cervical mucous membrane on an applicator. Personally I prefer to use carbolic acid because it is a little less likely to produce complicating constitutional symptoms. If you are able to get through the cervical canal and apply pure carbolic acid to the mucous membrane this will prove an effectual local anesthetic.

The tampon of glycerin that is applied after the dilatation continues the stimulation of the circulation. The effect of the glycerin is to draw serum from the blood. This causes an increased flow of blood to the part and proves an excellent stimulant to the circulation. The affinity of glycerin for moisture is well known, and it may be used at any time as an excellent mechanical means for the unloading of congestion of the cervical tissues.

Of course, for from 10 or 12 to 24 hours after the dilatation and insertion of the tampon there will be a free discharge. To prevent discomfort and uncleanliness a napkin will have to be worn. The tampon is left in place, as I have said, for 36 hours. Its removal is followed by a hot douche which should be repeated daily. The treatments are continued twice a week for periods of three weeks, being suspended during the menstruation week. The series of treatments continue for two or three months. Usually the patient is encouraged by having the first menstruation that follows the beginning of treatment a source of much less discomfort than before. Not infrequently it is found that patients come back during the third or fourth month to give the symptoms of physiologic suppression of menstruation and beginning pregnancy.

In cases in which the condition has been a source of discomfort for several years after marriage and it is evident that there is degeneration of the mucous membrane lining the uterine cavity, dilatation should be followed by thorough curettage. After curettage the interior of the uterus should be swabbed with pure carbolic acid and the cavity packed with gauze. There is considerable difference of opinion as to whether the uterus should be packed with gauze in these cases, a drainage plug inserted, or left to drain as best it may. Personally, as the result of 20 years' experience, I prefer to use the gauze packing. It acts as a foreign body and stimulates the uterus to contraction. This is an excellent feature of the treatment for the uterine muscle. Every muscle must be exercised in order to develop properly. The uterine contractions tend to force out the gauze, a purpose that may be accomplished or may not, but in any case the object of enforced muscular function is attained.

Vigorous contractions of the uterus may expel the gauze on the second or the third day. At times on the fourth day, when its removal from the vagina is being accomplished, it is found that none is left in the uterus. The gauze should always be taken out not later than the fourth day.

After the gauze is removed the uterus should be swabbed out once more with pure carbolic acid and then hot vaginal douches should be employed once a day for at least 10 days. The 4 days of irritation and contraction are usually enough to start the uterus on its career of development.

When the patients are unmarried women, it seems better always to employ an anesthetic. It is a disagreeable task to have to treat a young unmarried woman, or to make frequent examinations and regular dilatation of her external genitals, such as is outlined in my remarks with regard to young married women. Dilatation should be accomplished at one sitting, and then the vaginal tampon and douche treatment should be employed only in case there is not a prompt reaction that causes menstruation to be much less uncomfortable and more abundant during the months that follow the surgical treatment.

Of course, during these local therapeutic measures the general health of the patient needs to be particularly cared for. Outdoor exercise must be insisted on, and in general every precaution taken, so that the patient's constitution is in a good condition to lend itself to the special efforts required for uterine and genital development.

I have been asked whether electricity is good for infantile uterus. There is no doubt that electricity properly applied may be a valuable agent for stimulating the uterine muscle and causing it to develop. As a rule, however, electricity is used by men who are not able to diagnose the pathologic condition that is really present, and whose results are therefore not to be considered seriously as representing the curative effects of the electric current. There is no doubt that electricity may do serious harm if used when inflammatory conditions are present. Employed, as it usually is, by men who understand the physics of electricity but not the diagnostics of gynecology, harm may result. Unfortunately it is mainly used by quacks who make exaggerated claims for its therapeutic properties.

Some years ago, it will be remembered, the use of electricity for the treatment of fibroid tumors of the uterus became a very common practice. The system of treatment obtained its vogue because of the reports of results obtained by Apostoli, of Paris. He announced that in some cases small fibroid tumors of the uterus could be made to disappear, while in larger tumors the symptoms could be relieved simply by the proper use of electricity. Most prominent American gynecologists thought it worth the while to obtain a battery and the necessary instruments in order to be able to use electricity on such patients, sufferers from fibroid disease, as refused operative intervention.

In order to learn the details of the technic, I went to Paris and studied under Apostoli. I found that some of the patients who were suffering with fibroid tumors had been under his treatment for 20 years, and while they were supposed to be relieved of their symptoms, many of them still found it necessary to continue the treatment. On the other hand, I found that Apostoli himself was a very poor gynecologic diagnostician. He frequently found tumors where, after the most careful examination, I could not detect any, and I found it easy to understand how, in such cases, he could eventually report the disappearance, or at least the absence, of fibroid tumors. I did not see a single case in which I could be assured in any way that a fibroid tumor had been dissipated by electrical treatment.

Some five years ago, as secretary of the American Gynecological Society, I suggested that there should be a discussion of the subject of the use of electricity in gynecology. In this discussion many of the prominent gynecologists of this country took part. The conclusion arrived at with practical unanimity was that the benefits to be derived from electrical treatment in gynecology are mainly mythical. Many of the members of the Gynecological Association had provided themselves with batteries and a complete set of paraphernalia for electrical treatment, but they had either relegated them to the cellar or were waiting to sell them at a sacrifice and be glad to have them taken off their hands.

I may confess that in Paris I bought an expensive French electrical outfit before attending Apostoli's clinic, and that I brought it home to America with me, but I have never used it. My experience is in accord with that of most of the gynecologists of America, certainly all of those who have reached any prominence in their chosen specialty. You will hear much of the benefits to be derived from electrical treatment, but always from men whose fad is electricity and who are not known for their ability as operators or diagnosticians.

Besides infantile uterus, there is another condition that is a very common cause of sterility. This second cause consists of adhesions of inflammatory character that occur either in the tube or around the ovaries. The ovaries may become surrounded by a false membrane of inflammatory exudate, thin and transparent in character, yet absolutely barring the entrance of the spermatozoa or the extrusion of the ripened ovum. One of the great revelations that has come as the result of Lawson Tait's development of gynecology is the fact that even slight inflammatory adhesions involving the tubes and ovaries, conditions which give no symptoms or but very slight manifestations of their presence, may yet completely cripple the function of the tubes and ovaries. Any one who is familiar with the anatomy of the uterine appendages and knows what delicate structures they are, will readily realize how facile is their closure by a pathologic condition. The fimbriated ends of the tubes, for instance, may very easily become adherent as the result of an inflammation that produces scarcely any noticeable changes and yet causes absolute preclusion of all possibility of conception.

No amount of stimulation of the circulation will affect this pathologic condition. The only treatment that proves successful is to break up the adhesions, making the tube patulous once more and leaving the surface of the ovary uncovered. For this it may be necessary to enter the peritoneal cavity, but even this serious objection is justified when there is proper hope of putting an end to the sterility. The breaking up of such adhesions through a vaginal incision is not difficult, and as this operation is practically without mortality, the procedure represents one of the most practical advances in modern gynecology.

I distinctly remember one of my first cases, rather more serious in the condition found than is usually noted. The left ovary was removed because of an abscess in it; the left tube because of a pyosalpinx. The right ovary was resected, there being a slight inflammatory condition of one pole, and the fimbriated end of the tube was carefully opened up. The round ligaments were shortened in order to overcome a tendency to retroflexion of the uterus. The patient was completely relieved of her symptoms, and a year and a half later gave birth to a child. Since then her uterus has been in good condition. I have since had many similar cases.

There is no doubt in my mind, then, that the surgeon is perfectly justified in opening the abdomen, at least through the vagina, in order to remove pathologic conditions that prevent conception. Patients do not suffer from the operation itself, and have practically no discomfort afterward, only being kept in bed for some two to three weeks. When other treatment has failed I consider that an exploratory incision for the purpose of determining, if possible, the cause of the sterility is perfectly justified. At the meeting of the American Medical Association, at Saratoga, in 1902, I gave the details of 4 cases, patients operated upon simply for the relief of sterility. Of these patients, 3 have since given birth to children.

The most common condition found is an occlusion of the tube. When this is present it is not always necessary, or even advisable, to resect the tube. It may only be necessary to render it patulous once more. Chronic gonorrhea is the usual cause of the occlusion of the tube, but in time the gonorrheal secretion may become dried up and sterile. It is not an unusual thing to find that small lumps of dried material may by gentle massage be forced out of the tube. When warm salt solution is sprayed upon the tube and gentle massage is done at the same time, the organ will be rendered thoroughly patulous, without any danger of infecting the peritoneum, since the manipulations are carried on in the vagina. At times a simple hydrosalpinx is found and the mucous membrane of a distended tube may, under these circumstances, prove perfectly healthy. After determining by means of the probe that the tube is patulous, all inflammatory false membranes around the ovaries should be stripped off so as to make the condition favorable for extension of the ova.

There is great gratification in this work for the gynecologist, for he finds that while it is often said that American women do not care to have children, yet many patients frankly come and say that it is not so much relief of their symptoms that they want as relief of their sterility. Every woman should be told, after the competency of the husband has been determined, that her sterility may be due either to infantile uterus or to inflammatory adhesions; but none of them must be encouraged to think that an immediate cure is possible. There is no doubt that many married women who are now unhappy because of lack of children may be consoled by conservative gynecology, and that, too, without being subjected to any serious risk.

ECTOPIC GESTATION, WITH NOTES ON A CASE OF TUBAL MOLE

A PAPER READ AT A MEETING OF THE WIMBLEDON MEDICAL SOCIETY

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Misplaced or aberrant gestation with its synonyms, Conceptio vitiosa, Grossesse par erreur de lieu, etc., is mentioned in the writings of an Arabian physician—Albucasis—as far back as the eleventh century. In this case fetal bones were withdrawn through a suppurating sinus at the umbilicus. Other cases were published during the fifteenth and sixteenth centuries. Primrose records a case in which ectopic gestation was diagnosed on the left side of a patient's abdomen in 1591. This was followed by signs of a similar occurrence on the right side in 1594. The right-sided fetus was discharged in 1595, piecemeal, through a suppurating sinus, whilst the fetus on the left side was removed by operation, the patient being restored to health.[1]

The first time that the Fallopian tube is mentioned as containing a fetus is in 1604. It is mentioned again in 1638, in which year Riolan records cases. The early history of this condition is not confined to women, inasmuch as the disaster was proved, as long ago as 1597, to occur in the cow, and later instances are recorded in the case of sheep, bitches, hares, etc. The term "ectopic" was first introduced by Robert Barnes; and it seems in every way suitable, since it is sufficiently comprehensive to include all misplaced gestations whether the sac forms outside the uterus, in the wall of the uterus, or even in the lower zone (placenta prævia)—but the last condition cannot be brought within the limits of this paper.

Extra-uterine gestation as a subject has been divided into varieties, the number of which has varied much from time to time. Until

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[1] W. Campbell, Extra-uterine Gestation, p. 6.

1824 three varieties—tubal, ovarian, and abnormal—held the field; then came, in 1837, the laborious tenfold division of Dezeimerie. The latest classification is that of Lawson Tait's, in which he puts the subject under three headings: (1) ovarian, possible but not yet proved (1888); (2) tubal; and (3) tubo-uterine.

In the year 1899 ovarian pregnancy was proved to be a fact by Dr. Catherine van Tussenbroek, of Amsterdam,[1] and in January, 1901, Anning and Littlewood showed a specimen at the Obstetrical Society of London which settled the question of ovarian pregnancy once for all. It is now an established fact that an early embryo-sac has been seen inside an ovary in microscopic section. Mr. Bland Sutton, on the memorable first Wednesday in January, 1901, came to the Obstetrical Society's meeting with sections in his pocket of van Tussenbroek's specimen, to obtain which he had made a special pilgrimage to Amsterdam the week before.

This specimen satisfied the postulate laid down by himself that, to prove the case, " an early embryo in its membrane, contained in a sac in the ovary, should be forthcoming," for as he and Tait, and indeed Velpeau and Allen Thompson years before had pointed out, nothing could be argued from large gestation sacs surrounded by matted and adherent tissues. No one now has reason to doubt the occurrence of ovarian pregnancy, and in the light of present knowledge we may even yet accept the view expressed by Campbell, in 1840, that " ovarian gestation" is not so rare as it was at one time supposed to be, for the chances are that many ovarian moles have been missed, as their true nature could only be disclosed by the microscopic discovery of villi in the clot; and one hears that Mr. Bland Sutton, with characteristic enthusiasm, is having every ovary containing a blood-clot carefully examined for gestation products. How the event of ovarian pregnancy occurs, whether it is essential for the fimbriæ of the tube to be adherent to the surface of the ovary, as was the case in Anning's specimen, so that the rupture of a follicle of de Graaf occurring within the area embraced by the fimbriæ, and so exposing the contained ovum to the influence of a spermatozoon lurking in the neighborhood of the ostium, is a matter not yet settled; but I can scarcely believe that the male cell has the power of penetrating the coats of the ovary and so entering an unruptured follicle. On the other hand, such a combination of circumstances as I have suggested must be extremely rare; and if this is the correct etiological view, we must not expect to find gravid ovaries figuring as common museum objects in the future. Before leaving the subject of primary ovarian gestation, mention may be made of the case published by Hastings Gilford [1] as an example of this condition. The authenticity of this specimen was confirmed by a special pathological committee appointed to examine it by the Obstetrical Society of London, January 16, 1901. Up to the present time, therefore, we have the details of three cases only of ovarian pregnancy—one from Amsterdam, the other two recorded in London. In Condamin's case [2] the proof falls short in the most essential detail—ovarian tissue was not found to surround the gestation sac.

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[1] Annales de Gynécologie, December, 1899.

The third variety in Tait's classification only comprises a rare pathological disaster. I refer to the condition of tubo-uterine gestation. This is sometimes called interstitial pregnancy, although Doran points out objections to the term. The sac occupies that part of the tube which is embraced by uterine tissue. During his curatorship of the Museum of the College of Surgeons, Mr. Doran examined and reported upon one case of tubo-uterine gestation, and in searching through the museums of the London hospitals he found five other specimens. Parry before this, in 1876, recorded 31 cases in a series of 500 ectopic gestations he had collected; but it has since been proved that many cases thought to be tubo-uterine turned out to be cornual pregnancy, and since Doran's list was made very few authentic instances are on record.

This is fortunate from the patient's point of view, for until recently I have never read of a patient that recovered. The sac usually ruptures into the peritoneal cavity sometime between the sixth and twentieth weeks and with uniformly fatal results. Lapthorn Smith,[3] a Canadian surgeon, is reported to have successfully operated for ruptured tubo-uterine pregnancy. The crisis happened six weeks after the last period, and one gallon of fluid blood is said to have been removed from the abdominal cavity. Bland Sutton says that the rupture may occur into the uterus and the fetus be discharged per vaginam, but I have not succeeded in finding any case in which this occurrence has been proved to have taken place.

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[1] Lancet, June 24, 1899.

[2] Lyon Méd., March 2, 1902.

[3] British Medical Journal, April 5, 1902, Epitome.

Grun [1] showed a case of supposed extra-uterine gestation with birth through the uterus in which the uterus was not bicornuate, nor was there any hernial pouch; but no mention is made of the tube and, excepting that the right-sided tumor disappeared, the evidence of the right tube having been the seat of gestation rested purely on observations made at the time of delivery. Therefore no great value can be placed upon this case, the woman having recovered and no specimen being forthcoming.

The second variety mentioned by Lawson Tait is tubal gestation, by which is meant pregnancy occurring anywhere in the free portion of the Fallopian tube. This class comprises nearly every case of extra-uterine fetation. Lawson Tait put the whole study of ectopic gestation on a new footing in his epoch-making brochure written in 1888, in which (page 15) he says, " I believe they (ectopic gestations) are really all tubal, and in this consists the novelty of my views on the subject." Previously to this one sees again and again recorded in the Transactions of the Obstetrical Society examples of " undoubted" primary abdominal pregnancies which, through Tait's teaching, we now recognize to be examples of implantation subsequent to intraperitoneal rupture of the tube. There is no belief at present in primary abdominal pregnancy, though to my mind such an occurrence would be no more wonderful than the already proved ovarian variety; but since a rupture of a tube may heal completely so as not to be noticed, and since the abdominal ostium will assume its normal appearances after complete abortion, the contention that all abdominal pregnancies are primarily tubal must remain incontrovertible.

There is yet one position for the aberrant gestation sac not previously mentioned, and this is undoubtedly a secondary position which can only be assumed after rupture of the tube through its non-peritoneal surface, *i.e.,* into the mesosalpinx. Here the ovum passes out into the extraperitoneal cellular tissue which exists between the layers of the broad ligament, where its future course will be referred to later on.

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[1] Obst. Trans., vol. xxvii, 1885, p. 226.

As to the causation of misplaced gestation, we find that nothing whatever is known about it, and, as Bland Sutton says, "our ignorance will continue until we have some trustworthy information concerning the situation in the genital passages where the ovum and spermatozoon normally meet." The old obstructive theory which was largely believed in by many leading fellows of the Obstetrical Society of London until a comparatively recent date has fallen to the ground, since it is now well known that the ovum is more frequently retained in the outer and larger portion of the canal of the Fallopian tube than in its narrower and inner part.

Virchow was among the first to draw attention to the concurrence of perimetritis and parametritis with this disease, and thought that they stood in the relation of cause and effect. Lawson Tait held strongly that desquamative salpingitis, by robbing the tubal mucosa of its ciliated epithelium, removed the natural obstacle to the passage of spermatozoa and put the mucous lining of the tube in a condition similar to that of the uterus, so that fertilization of an ovum and immediate adhesion can then take place.

Tait's statement that the uterus alone is the normal seat of impregnation is indeed a pure assumption; but if we are prepared to adopt it, it is a theory which renders the pathology of ectopic gestation simple and intelligible—helping us to interpret the meaning of the period of sterility, or, in the words of Parry, "the previous inaptitude for conception," which precedes the occurrence of tubal gestation. During the past 18 months I have had the opportunity of examining minutely six specimens of tubal gestation. Three were removed by Dr. Amand Routh, one by Mr. Doran, one by Dr. McCann assisted by myself, and the last is my own specimen.

In Dr. Routh's three cases there was no evidence of pre-existing disease of the adnexa. He describes the opposite tubes as healthy as seen at the operation, and in the ovaries and tubes which I have examined there was no proof of the existence of previous inflammation. In Mr. Doran's specimen, I received both tubes, and these were undoubtedly the seat of long-standing salpingitis—each being enlarged to the size of an index-finger, and, as the result of kinking due to old adhesions, they were very sacculated; indeed, the non-gravid tube was so matted to the ovary at its distal end that the fimbriæ were no longer distinguishable. Dr. McCann's case was complicated by a cystic ovary on the same side and a hydrosalpinx on the opposite side. In my own case there were signs of old pelvic inflammation and the opposite tube was dilated and bound down by adhesions. A striking fact in this small series of cases is that the non-inflammatory cases all ended in rupture, whereas the others terminated in moles.

Converlaire, of Paris, last year wrote a very important monograph on the pathology of tubal pregnancy. He thinks the desquamative salpingitis of Lawson Tait to be purely imaginary as far as concerns the question of ectopic gestation. More recently, Petersen, of Copenhagen, in a thesis published this year on the evidence of 14 cases, supports the older view that inflammatory changes are at least predisposing, if not exciting, causes of tubal moles. Clarence Webster holds that the stimulus of impregnation first sets up a decidual reaction which, when it extends as an abnormal condition to the tubal mucosa, arrests the ovum, and thus causes the pregnancy to develop in the tube. Converlaire will not accept the existence of such a reaction, and there is nothing analogous in the gravid tube to the formation of a maternal decidua as seen in gestation in utero. My own investigations on the effect of the presence of an ovum in the tube upon the tube itself go to show that, far from the mucosa swelling up or becoming in any way actively changed, the plicæ in contact with the ovum show a process of obliteration, and appear as fibrous strands supporting degenerated cubical epithelium. These soon disappear, leaving the nude muscle of the tube wall exposed to the action of penetrating villi; cells and buds form and the latter dissect up the muscle-planes, and aided by interstitial hemorrhages complete the work of separation and thinning of the muscle walls until nothing is left but the peritoneal coat. When the tension of the intratubal contents exceeds the limit of the elasticity of the serosa, the latter yields and the tubal rupture is complete.

The results of aberrant pregnancy are, (1) rupture into the cœlomic space or into the broad ligament; (2) tubal abortion; and (3) the formation of a tubal mole by hemorrhage into the subchorionic chamber from the fetal circulation, the clot so formed remaining quiescent in the tube.

Rupture of a gestation sac mav be primary, that is, tubal rupture; or secondary, that is, rupture of the adventitious sac formed around a successful implantation within the layers of the broad ligament.

Primary rupture may be intraperitoneal or extraperitoneal. It occurs from the third to the tenth week. The result of intraperitoneal rupture is that the ovum, together with a variable quantity of blood, escapes into the cœlom. If rupture occurs after the first month, the hemorrhage is often violent enough to cause death in a few hours; but in earlier rupture the loss may be gradual, and the blood will clot, producing a so-called hematocele (Nélaton): this may become absorbed, or it may suppurate, or give way with a fresh hemorrhage.

The embryo which has escaped into the abdominal cavity, according to Sutton, invariably dies; but Tait[1] says "what have been called abdominal pregnancies are clearly exceptional cases where primary tubal rupture at the end of the third month has not proved fatal, where the extruded placenta has made for itself visceral attachments wherever it has touched, or where secondary rupture of a broad-ligament cyst has converted an extraperitoneal ectopic gestation into one within the peritoneal cavity." He says further, "that the first of these processes is by far the most common condition has been proved to me beyond doubt in my operations, for I have seen the ruptured tube within a few days of the catastrophe containing the great bulk of the placenta, whilst the villi of the extruded portion have been engaged in making epiphytic inroads on the intestine, the bladder, the back of the uterus, and the folds of the omentum. I have pulled these villi out of the living crypts they have made much as one pulls a barnacle out of its bed, leaving bleeding holes behind them!" An instance of the anterior abdominal wall being the site of a placental attachment is to be found in the Transactions of the Obstetrical Society of London, and another of a free amniotic sac containing a fetus at full term. Both cases giving support to Tait's, rather than to Bland Sutton's, views on this point. The result of primary extraperitoneal rupture is more favorable for the mother, as the hemorrhage is never so excessive between the layers of the broad ligament as it is in cases in which the blood escapes into the free peritoneal cavity.

After a sudden hemorrhage, the ovum gradually (according to Sutton) makes its way, as the rupture enlarges, into the mesometric tissue, so that it at no time loses its maternal attachment, and hence it continues to live and grow within the mesometrium. The sac so formed may rupture at any time between the twelfth week and term.

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[1] Lectures on Ectopic Gestation, 1888, p. 13.

Secondary rupture is where the above mesometric gestation sac has given way. The effects of this will vary according to the position of the placenta. If it is lying above the child in the upper part of the sac, it will be implicated, and fatal hemorrhage is the usual consequence beyond the mid-period of pregnancy; earlier the hemorrhage is less severe. If the fetus is uppermost, it will escape through the rent and be found lying (as in Jessop's case[1] in the Leeds Infirmary) free among the intestines, but still alive.

Tubal abortion can only happen during the first two months, for by this time the ostium is hermetically sealed by changes in the tube consequent on gestation. Tubal abortion is more often incomplete than complete. This is the great cause of pelvic hematoceles, a condition which most authorities now regard as caused by rupture or abortion of a gravid tube, although W. H. Freund and others have cited cases as evidence to the contrary.[2] Veit believes that only 27 per cent. of hematoceles are due to ectopic gestation. This condition is dangerous from the fact of its incompleteness—the hemorrhage being recurrent so long as the mole is retained in the tube. If a tube does not abort, it will rupture, or more rarely a quiescent mole will form.

Mr. Bland Sutton, in 1899, was the first to demonstrate the existence of tubal moles. He stated that moles only arise in the first few weeks following fertilization; that they are usually extruded through the ostium or through a rupture; and that they are by many considered impossible for diagnosis before rupture. In three of the six specimens I have mounted myself the tubes which contain moles are unruptured, and in two of the cases, at least, a correct diagnosis was made before operation. Mr. Bland Sutton himself showed a large tubal mole within an unruptured tube at the Obstetrical Society of London, in February, 1901. From this evidence, I am inclined to think that a quiescent tubal mole is fortunately not so rare as it is represented to be.

Signs, Symptoms, and Diagnosis of Ectopic Gestation.—Under this heading we have first to note that we cannot exclude ectopic gestation because the usual signs of pregnancy are wanting. The history most suggestive of aberrant pregnancy is that there has been a long term of sterility during which the periods have been regular; then a period is missed; pelvic pain is complained of, usually on one or other side, and on examination an enlarged tube is detected.

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[1] Trans. Obstet. Soc., London, vol. xviii, p. 261.

[2] Kober, Centralb. für Gynäk., No. 39, 1901.

Mr. Handley, one of my colleagues at the Samaritan Hospital, had the good fortune to watch the formation of a tubal mole in a woman giving a history somewhat as I have indicated. On the first examination there was no definite enlargement of the suspected tube, but seven days later a distinct swelling had formed. This was removed before rupture had taken place.

The rupture is signalized by unmistakable signs of internal bleeding—shock, anemia, syncope, etc.—which vary in degree according as the tube has ruptured into the cœlom or into the mesometrium. The suddenness of acute symptoms and a sense of something having given way, the woman having stretched for some object beyond her reach or exerted herself by vigorous bodily exercise, such as cycling, are characteristic. The patient may never recover from the initial syncope, as in the case of the English actress who died suddenly whilst swallowing an ice in a café in Paris. Such acute symptoms have to be diagnosed from those due to perforating ulcer of the stomach or of the vermiform appendix, acute obstruction of the intestines, twisted ovarian or dermoid pedicle, the passage of gall-stones, and renal calculi. As the shock passes off, the patient may continue too ill to leave her bed for some weeks, and the next thing to appear is a pinkish vaginal discharge accompanied by shreds or the passage of an unmistakable decidual membrane. If only shreds come away, examine them with the microscope; if they contain villi, the case is one of uterine gestation.

After recovery from rupture, the menstrual function is disturbed sometimes in the direction of amenorrhea; more frequently there is metrorrhagia for some considerable time, the flow coming from a uterus which contains, or has contained, a decidual membrane, and even if empty is sympathetically congested and enlarged. The cervix is soft and the os patulous; it may be pushed forward by a mass in the posterior pelvis. This mass can be observed to increase in size week by week, and, as a rule, cannot be separated from the uterus. If a crisis supervenes again, it indicates secondary rupture.

Fetal heart-sounds may be heard; placental bruit and fetal movements may be easily distinguished in some cases. Spurious labor sets in at term, the bearing down, as a rule, producing no result save the passage of blood and mucus from a dilated cervix; but occasionally the sac may rupture. This all subsides, and if milk has been present in the breasts, it will gradually dry up; the abdominal swelling shrinks, all signs of fetal life disappear, and suppuration in the sac with decomposition of the fetus follows, with sinus formation and the passage of fetal bones.

Among the conditions to be distinguished from tubal pregnancy are, pregnancy in a horn of a uterus bicornis,[1] retroversion of a pregnant uterus, ovarian and paraovarian tumors, growths in the broad ligament, hydrosalpinx and pyosalpinx, ovarian abscess, fibroid tumors. Tubal pregnancy may be concurrent with ovarian tumor, uterine pregnancy, and the presence of growth in the pelvis, and each tube may be the seat of a gestation sac contemporaneously.

Treatment.—If the condition can be diagnosed before rupture or abortion, laparotomy should be performed and the gravid tube removed. For symptoms of internal hemorrhage, we have simply to apply the common surgical rule for treatment of primary hemorrhage, and cut down, define, and tie the bleeding point, remove the injured tube, and, if the rent extends into the fundus, carefully sew it up with chromicized catgut, using a double layer if necessary.[2] After acute symptoms have subsided and a mole or hematocele has resulted, there is a tendency of late to adopt a conservative line of treatment and leave things to nature, subjecting the patient to very prolonged rest in bed. Krönig[3] compares operative results with conservative treatment, and concludes strongly in favor of the latter.[4]

Mesometric gestation produced by primary extraperitoneal rupture will call for interference if the child continues to grow. Up to the fourth month the parts may be removed en masse; later on we have to consider the treatment as applied to the sac and to the placenta. The removal of the sac entire was once done successfully, but the proceeding is too dangerous to render the attempt justifiable. It is safer, after the removal of the fetus, to stitch the edges of the sac to the abdominal wound and drain.

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[1] Centralb. für Gynäk., No. 2, 1902.

[2] Lapthorn Smith. Montreal Medical Journal, August, 1901.

[3] Centralb. für Gynäk., No. 42, 1901.

[4] See also Conservative Treatment of Extra-uterine Pregnancy, by the author. Journal of Obstetrics and Gynecology of the British Empire, June, 1902.

With regard to the placenta, if it is situated above the fetus, an attempt should be made to remove it. When the placenta is below the fetus and the latter is alive at the time of operation, leave it after cutting off the cord, and then sew up the sac. If the child be dead, its placenta can be removed without risk. I remember seeing a case in which a placenta was left in a suppurating sac after removal of a fetid fetus; the end of that case was fecal fistula, emaciation, and death. Do not wait until an extra-uterine child is viable before operating, as the mother runs a perpetual risk of secondary rupture, and the child is generally not worth saving. After the death of the child and the formation of sinuses, enlarge these and remove every bit of bone, or the sinus will persist.

The following are the details of a case of unruptured tubal molar pregnancy:

C. O., a married woman, was sent to me, May 3, 1901, by Dr. Kennedy, of Plaistow, as a case of ectopic gestation. The patient gave me the following account of herself: She had been 10 years married, and had had two children and two abortions, the first in 1897, and the last in February, 1901. Menstruation was regular until December, 1900, when it ceased. In February, 1901, she began to suffer from sharp pelvic pains, sickness, and feelings of faintness. For this she stayed in bed for three days, and whilst lying up lost blood in small quantities. It was at this time that she had what she called her second miscarriage, as something "formed" came away; fluid made its appearance in the breasts at the same time. After lying in bed for three weeks, the patient got up, but continued to lose blood until the middle of April, when the flow stopped for a fortnight. When she came to me on May 3, she had been losing for five days. Prior to seeing me she had been in bed for three weeks with pelvic pain and dysuria, especially at the end of micturition. The bowels had been regular, there had been no blood in the motions.

On examination there was a swelling in the hypogastrium somewhat to the right of the midline, and there was milk in the breasts. There was no discoloration of the vaginal mucous membrane; the fornices were free and the os patulous. The tumor and uterus appeared one; they moved together, any movement causing much pain. Her temperature was normal, and there was no albumin in the urine.

She was admitted to the hospital May 30, and was operated on the next day. On opening the abdomen, a large rounded mass rather larger than a closed fist was seen on the right side, rising up out of the true into the false pelvis and covered by adherent omentum. The mass capped the uterus, having the same relation to it as an adrenal has to a kidney, fitting itself to the fundus, and overlapping it behind and in front. The mesometrium was free on this side. It was ligatured and cut away from the tumor, the latter having been peeled off the uterus and the tube divided and tied. The mass was the right Fallopian tube distended by a mole. The left tube was adherent to the ovary by inflammatory bands which were broken down, but nothing on this side was removed. The fundus uteri was sewn to the anterior abdominal wall and the latter united by three layers of silk. The patient made an uninterrupted recovery and left the hospital on June 19, 1901. She came up again recently, and the house surgeon reports her as being in excellent health.

Many would criticise the removal of a tubal mole of this description, but the fact that the woman was a chronic invalid before operation and is now in excellent health amply justifies the procedure.

THE SIGNIFICANCE AND SOME SUGGESTIONS REGARDING THE TREATMENT OF CERVICAL LACERATIONS

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A LACERATION of the cervix uteri, occurring as it does almost invariably during parturition, has a greater significance than would appear at first sight. It is an accident so often associated with other conditions, and giving rise to, or followed by, serious morbid changes in and about the pelvic structures, that its importance calls for more than ordinary attention and skill.

A larger proportion of parturient women meet with this accident to some degree in their first parturition than subsequently; but, fortunately, the tear in many cases is slight, and the recuperative powers of nature then are so active that no untoward results are noticeable. As a rule, the accident is not known at the time to have occurred, unless it is very deep,—to and within the vaginal vault,—when, in consequence, an unusual post-partum hemorrhage follows. Even then it may not be detected, for the infravaginal cervix and the vaginal vault are not inspected, to disclose the arterial issue of blood from the ruptured circular artery of the cervix. A parturient woman may die from loss of blood post-partum, coming from this vessel, or a lacerated bulb of the vulvar vestibule, notwithstanding the gravid uterus is well emptied and firmly contracted.

Should, now, from any cause, this tear be neglected, a quite vascular and very susceptible portion of the uterus is made bare, for the absorption, through open vessels and exposed lymphatics, of any septic germs from within the uterus above or the vaginal tube below.

Almost every parturient woman, in the event of an injury of this kind to the cervix uteri, suffers to some degree with septic absorption, and an inflammation of the base of the corresponding broad ligament, as well as of the endometrium. This is the underlying causative factor in most cases of so-called pelvic cellulitis,—largely an obstetrical disease.

Nor are these results all. Any septic inflammation of the genital tract of a lying-in woman delays involution of those parts. A torn and imperfectly uniting cervix uteri leads to subinvolution of the whole uterus, with, of course, some septic endometritis and metritis. The cervix soon looks eroded, becomes everted, takes on a granular degeneration and a cystic formation. Its parenchymatous structure becomes hyperplastic, indurated, elongated, and enlarged. So, in consequence, this central organ of her pelvis becomes displaced, first downward, then backward. Retroflexion of the uterus is often conjoined with retroversion. Fortunate, indeed, will this woman be, if she escapes a resulting salpingitis, with destructive alterations in the structure of the periuterine appendages.

Naught need be mentioned of a persistent post-partum bloody flow, the exhausting menorrhagia and metrorrhagia, and the troublesome leucorrhea, followed it may be by a permanent sterility. How many women have been broken down in general health, or made constant sufferers, from some functional nerve disorder, reflected from this seemingly insignificant local lesion of the cervix uteri ?

This picture is not overdrawn, in depicting all of these aforesaid consequences. They happen; not, of course, always after lacerations of the neck of the womb, but they do happen often enough to make the attentive physician anxious as to their possibility.

There is no local lesion of the genital tract in women which, when severe and bilateral, or multiple, is directly followed by so many local morbid changes, and so much general reflex disturbance, as is the one under present consideration. Cervical lacerations, then, signify much.

Probably there is no one piece of work which justly entitles the name of Thomas Addis Emmet to a world-wide reputation and an everlasting earthly renown, as does his contribution to practical gynecology, in this very particular. To this man are we indebted for the original recognition of the exact cause and nature of this common lesion of the cervix uteri, and for his practical suggestions and executions of treatment, in its immediate and complete relief.

It is reasonably certain that this injury of the cervix uteri does not occur as frequently as it did several decades since—another evidence of a better obstetric management now of women in labor.

Nature does the best she can to make repairs for these lacerations, by filling up the gaps with new connective tissue, organized from the granulations thrown out. But cicatricial tissue is a repair tissue, and is always inferior in kind to the natural structure. Cicatricial tissue has a low grade of vitality, is sometimes unduly sensitive to touch, is a source of local irritation, and quickly becomes a seat of reflex irritations; finally, it is prone to take on certain degenerative changes in the declining years of life.

The causal relationship existing between lacerations of the cervix and malignant disease of the uterus is now a matter of almost universal knowledge, and should be recognized and borne in mind by all physicians. Excepting the mammary gland, the second most common seat of malignant disease in women, the uterus is five times more frequently involved than is any other organ in women. What, now, are the facts about the matter? (*a*) Carcinoma in women is found most frequently in the uterus. (*b*) Most cases of carcinoma of the uterus involve the cervix. (*c*) Almost all of the cases are observed in parous women, and in that portion of the uterus most exposed to injury during parturition—the cervix. Carcinoma rarely involves other portions of the uterus, and when it does occur in the fundus it is usually in nulliparæ.

Carcinoma of the cervix uteri, in the vast majority of instances, commences in the cicatricial plug, in the neighborhood of the os externum, where the squamous epithelium of the infravaginal cervix joins the cylindrical layer of epithelium within the cervical canal.

There is, of course, in cancerous disease of the cervix uteri, the stage of innocency, the transitional stage, and finally the stage of malignancy. Aside from the diagnostic evidences of the microscope, there is a reasonable inference, deducted from the results of some topical medication, as to the stages in which the disease presents itself.

While these views seem so reasonably certain, based as they are on the evidences mentioned, it ought not to pass without calling to mind that all of the recognized English authorities do not so coincide. For instance, C. Hubert Roberts, Physician to the Samaritan Hospital for Women, and Demonstrator at St. Bartholomew's Hospital, is the author of a work on "Gynecological Pathology," in which he says: "Tears of the cervix uteri are probably unimportant, have few symptoms, certainly no reflex ones; nor does there appear to be any relation between them and cancer." And Sir J. Williams, of London, in his work on "Cancer of the Uterus," says: "Lacerations of the cervix uteri play no part in the causation of cancer of the uterus." May we not now fairly remark that the coincidence of these two conditions is too frequent to be explained on mere chance? Lacerations, then, of the cervix uteri are unquestionably a source of local irritation, thereby strongly predisposing to some malignant disease of the same structures, and sooner or later, in a considerable proportion of cases, manifestations of that disease are distinctly apparent.

If physicians are to save women from the fatal result of cancer of the womb—her most formidable enemy—they must first prevent the oncoming of this disease by doing more than has been done to prevent laceration of the cervix during parturition.

Lacerations of the birth canal may unavoidably occur, but many such injuries can be prevented by allowing more time for these tissues to dilate in the first stage of labor. Once occurring, all can be repaired.

Is it not true that many lacerations of the cervix uteri are provoked by some injudicious use of the obsteric forceps, in time, force, or direction, or by an improper use of version of the fetus? Nature may and ought to be directed. She ought to be permitted to take her course in this first stage of parturition. Bearing-down efforts at this time, on the part of the woman, and undue haste, on the part of the physician, have done much harm in this direction.

If, now, any cervical laceration has taken place within its muscular substance, especially if so deep that the circular artery is severed, a primary union should be secured by an appropriate stitching, on the same principle of conduct that the accoucheur always repairs a torn perineum at the time of its rupture.

The cervix will be bruised less, and the presenting portion of the fetal body will more easily be directed into the axis of the obstetric canal, if the patient is placed, in the second stage of labor, on that lateral surface of her body toward which the presenting part of the fetus projects.

A primary tracheloplastic operation is quickly and easily done. Two Sims' specula are needed; catgut is a good suture material. Primary union is almost certain, if the vagina and cervix have been freely cleansed with hot water and aseptic gauze. But a secondary tracheloplastic operation is one of the more difficult plastic procedures. Failures to some degree are by no means uncommon. The aim, of course, should be to restore the cervix to a normal size and shape, with a normal os externum. The restoration of the parts to a natural symmetry and relation are the chief indications of success.

A sharp curettage of the whole uterine cavity is an imperative preliminary surgical step. The endometrium of the corporeal cavity in chronic cases is invariably changed, and curettement often removes much fungoid material. Done at the same time, with proper aseptic precautions, it affords relief to the secondary endometritis. No packing of the uterine cavity ought now to be done.

Considerable care is required not to make the external opening of the uterus too small. Such may create painful menstruation, obstruct the natural drainage of the cavity above, or be the cause of an acquired sterility.

A serious contraindication not unfrequently presents itself. For instance, the presence of pronounced periuterine exudation, marked evidences of an old salpingitis and pelvic peritonitis, with fixation of the uterus, though not complete, are manifest contraindications to the operation. These conditions ought first to be relieved by rest, by repeated saline purgation, by hot vaginal douches, and by topical applications of ichthyol and boroglycerid, followed, it may be, by a vaginal section (postuterine), possibly by an abdominal section. It is a dangerous procedure to draw the uterus down, to curette its cavity, and then denude and stitch its cervical walls, unless the periuterine spaces are free, or comparatively free, from any secondary inflammatory action. Too often it is that some otherwise clearly indicated intrapelvic operation is brought into disrepute because of some individual misapplication.

Almost every gynecologic operator in these cases must have observed at times cases in which there is much enlargement, thickening, and induration of the cervical walls, irregular in shape, with extensive degeneration of the Nabothian follicles. Such conditions ensue in time on multiple or stellate lacerations, long neglected. Due normal restoration of the infravaginal neck, and a proper symmetry of these structures can, in such cases, only be obtained by a complete removal of all cicatricial tissue, by a thorough exsection of both anterior and posterior lips, and a thorough stitching of their surfaces, after the manner of Schroeder's operation, or some modification thereof. All risks of a stenosed cervical canal are prevented by the timely use of a cervical glass or hard-rubber plug, retained best in position by a single silkworm suture, until the stitches are removed.

Tracheloplastic operations properly performed in suitable cases, with due regard to thorough asepsis, are among the most satisfactory in gynecology; for not only is the local mischief very much bettered in its primary lesion, but in its secondary changes. Few gynecologic operations are followed by a more pronounced benefit to the general health at large than is the correction of this evil.

The special points in this paper are:

(1) How to avoid the undue frequency of this obstetric accident.

(2) Importance of its early recognition at the time of its occurrence.

(3) Our opportunity to correct the accident at the time of its occurrence.

(4) The ultimate dangers of lacerations of the cervix uteri, especially as to carcinoma of the uterus.

(5) Frequency of imperfect results attending these plastic operations, by: (*a*) Want of normal symmetry of the cervix uteri; (*b*) artificial stenosis of the cervical canal; (*c*) want of a free and thorough exsection of all unhealthy cicatricial tissue at the bottom of the rent, and a consequent continuation of local and reflex disturbances.

It is not a matter of surprise that an operation so generally successful in overcoming local morbid conditions and in relieving such frequent and far-reaching after-effects should have been greatly abused. No one has appreciated these results of his arduous work in this direction more than Emmet himself. It is impossible altogether to prevent this. Almost all operations—gynecologic operations in particular—have been often abused, or done in inappropriate conditions, and when not needed.

Ophthalmology

SURGICAL INTERVENTION IN PARALYSIS OF THE OCULAR MUSCLES[1]

BY PROFESSOR EDMUND LANDOLT,

Of Paris

It is self-evident that there can be no question of surgical intervention in paralysis of an ocular muscle except in those cases in which treatment of the cause has been of no avail and in which a spontaneous cure can no longer be hoped for. It will, therefore, be unnecessary to cite the various ineffectual treatments employed in the cases herewith reported. All were cases in which paresis or paralysis of one or several of the ocular muscles had been definitely determined.

The problem which presents itself to the surgeon is this: to remedy, by means of operation, weakness of action (paresis) or complete loss of action (paralysis) of an ocular muscle, or, rather, the inconveniences that result to the patient from such conditions: diplopia and false projection when binocular vision is present, and at least deviation of the eye when binocular vision no longer exists.

One versed in the anatomy and physiology of the motor apparatus of the eye will seek the solution of this problem in *an operation that will augment the action of the weakened muscle.*

It is strange that in such cases recourse has been had to weakening the antagonist. The setting back of the muscles acting antagonistically to the weakened muscle is still considered the first operation to be tried. Sometimes an effort is made to increase the effect of the tenotomy by means of a suture, sometimes by advancing the paralyzed muscle, but tenotomy is always the foundation of the surgical intervention.

Do we not read in a widely known work on ophthalmology, from the pen of a distinguished writer, that the remedy indicated in case of paresis of the external rectus is tenotomy of the internal rectus of the same eye? But, having thus weakened the collaborator of the external rectus of the other eye, it becomes necessary to set back the latter muscle to the same extent; the internal rectus of the unaffected eye having then a too feeble antagonist, tenotomy of this muscle in turn becomes necessary. To sum up the matter, according to this writer, in paresis of a muscle acting in the horizontal plane, the ideal treatment would be to reduce the action of the other three muscles to an extent corresponding to the amount of paresis. A strange method of re-establishing muscular equilibrium!

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[1] Translated by Dr. Annina C. Rondinella, Philadelphia.

What would we say of a surgeon who, in order to remedy a paralysis of the extensor muscles of the forearm, would cut the tendons of the flexors, thus adding to the already paralytic weakness of the extensors a traumatic weakness of their antagonists?—In the case of the eyes, the consequences of such an act are even more grave.

The setting back of an internal rectus, for example, not only limits the nasal excursion of the eye, for which the patient is obliged to compensate by turning his head, but it also limits convergence, a function indispensable to binocular work. A tenotomy, however slight (and more especially a tenotomy of both internal recti), renders convergence absolutely insufficient, and creates a most troublesome crossed diplopia at a time when binocular vision is most necessary.[1]

But all treatises on ophthalmology, even those that discuss advancement of the paretic muscle, continue to uphold tenotomy of the antagonist, assigning first rank to this operation; in the opinion of these authors, advancement of the paretic muscle is but an aid, and serves only to increase the effect of the setting back of the antagonistic muscle. And yet it would seem that, upon considering a case of muscular paresis, the idea of giving the weakened muscle greater control over the globe by means of a more favorable insertion, should naturally present itself.

Let us take, for example, a case of *paresis of the external rectus.* There is *convergent strabismus* and *homonymous diplopia.* The muscle that formerly turned the eye as much as 50° toward the temple (as the homonymous muscle of the other eye still does) now turns it only 35°. The field of excursion has lost, on this side, 15°. Is there the slightest hope of increasing this field by a tenotomy of the internal rectus? Not the least!—It is not the fault of the internal rectus that the outward movement of the eye is restricted; but the external rectus, because of its paresis, has no longer the power to effect a more extended temporal rotation of the globe. This seems very obvious, and yet a large majority of ophthalmologists still believe that reducing the strength of one muscle is of direct avail to its antagonist, as if, under the impulse of a temporal rotation, the internal rectus would counteract the action of the external rectus.

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[1] I have recently seen a young man in despair because of such a diplopia following a tenotomy of the internal rectus done two years ago.

No, in the presence of *paresis of an external rectus,* we reason as follows: The action of this muscle is insufficient, and we are powerless to increase its strength, but we can use what it still has to better advantage, by giving to the muscle a more favorable insertion, —that is to say, by bringing it nearer to the corneal margin and thus increasing the extent of its power over the ocular globe. It is as if we should give to a too weak arm a longer lever to lift a weight.

This change of insertion—this *advancement*—may be reinforced by a shortening, that is to say, by the *resection* of more or less of the extremity of the muscle.

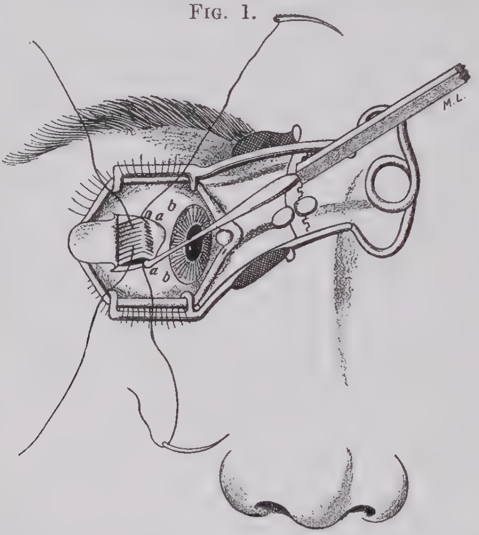
It may be said, in passing, that I have been perhaps the first to practise resection of a piece of the body of the muscle without touching its insertion on the globe, the two surfaces of the section being, of course, united by a suture—the muscle undergoing thus a shortening without advancement. The results of this procedure, without being bad, were however generally insufficient. It has, therefore, seemed to me that advancement of the insertion of the muscle is the operation to be commended, and that, if a resection is added, it is preferable to practise it at the tendinous extremity of the muscle rather than to sacrifice a part of its contractile tissue.

The operative procedure which I employ at the present time in muscle advancement is as follows:

Narcosis is indicated only in infants and unmanageable patients. All those upon whom I have operated without chloroform state that the operation is easily borne under simple instillations of cocain. Subconjunctival injections of cocain are not to be recommended, as they produce too great alteration of the tissues.

The speculum having been placed in position (Fig. 1), I seize a fold of the conjunctiva near the cornea and with scissors cut a flap as shown in the figure, the apex coinciding with the margin of the cornea, the base with the insertion of the muscle to be advanced. The apex of this flap is cut off, in order to avoid the swelling which the conjunctiva might form at the margin of the cornea. With a forceps, held in the right hand, I turn the globe in the direction opposite from the muscle to be advanced, and with a forceps held in the other hand I seize the tissue surrounding the muscle near the border in such a manner as to form a fold perpendicular to the direction of the muscle. I cut in it a buttonhole, sufficient to allow me to see the white sclera underneath. Only then can one hope to pick up on the hook, introduced through this opening, the entire muscle without its forming a fold, a requisite for a satisfactory advancement. We have, in fact, entirely given up the von Graefe method, which consists in introducing the muscle-hook by passing it between the conjunctiva and the muscle, the point directed at first upward, afterward turned while resting against the ocular globe and forced through the capsule of Tenon under the muscle. By this method, the muscle is folded, twisted, and often penetrated by the hook in such a way, that the sutures are badly introduced, the muscle is irregularly sectioned, and there is even risk of allowing some of its fibers to remain attached to the globe.

FIG. 1.



If the operator is not entirely satisfied with the position of the hook, if he fears, for instance, that the buttonhole has not been made sufficiently near the edge of the muscle, he may cut another one, corresponding to the blunt end of the hook, which comes out from the other side of the muscle, and he may there introduce another hook, which he pushes beyond the first slit and under the fibers which may have not been included upon the first hook, and the latter may then be withdrawn.

The hook is then entrusted to an assistant, who holds it without pulling upon the eye, drawing, however, lightly upon the muscle. The sutures are then introduced from without inward through the cellular tissue, the capsule of Tenon, and the muscle, one above and the other below, nearly to one-third the breadth of the muscle. The distance of the sutures from the insertion of the muscle depends upon the effect desired.

In a case of *simple advancement,* the sutures are placed in front of the hook not far from the insertion of the muscle, which is detached afterward close to the ocular globe.

If, however, greater effect is desired from the operation, *advancement combined with resection* of the muscle is indicated. In this case, the sutures are introduced behind the hook, as is shown in the figure, their distance from the insertion of the muscle depending upon the extent to be given to the resection.

The sutures being in position, one branch of a pair of scissors with a lateral curve is passed in front of these under the muscle, the scissors are then closed, and with one stroke the muscle is severed. The hook is now freed, and the tendinous end of the muscle is carefully detached from its insertion to the globe.

Now, seizing the four ends of the sutures with the left hand, the muscle is lightly raised, in order that the operator may assure himself that the threads are not too near the section surface, that no part of the muscle is adherent to the globe, and that it may be easily drawn toward the margin of the cornea. The extremity of the closed scissors, managed by the right hand like a muscle hook, is very useful in this procedure.

One has now only to proceed to the new attachment of the muscle. This is one of the most delicate parts of the operation. Indeed, the muscle with the surrounding tissue, which must always be included in the suture, offers resistance enough never to give way.

There is more danger of loosening of the suture at its point of attachment to the ocular globe. In order to find as much resistance as possible, I seize the conjunctiva and underlying episclera between the ends of my forceps, at the points *a* and *b* (Fig. 1), and I introduce the needle as deeply as possible into the episcleral tissue, without, however, passing through the sclera. The ease with which this is accomplished varies in different patients. In children the conjunctiva is sufficiently resistant and the episclera sufficiently soft to be easily penetrated by a sharp needle as far and as deep as the operator wishes. In adults, on the contrary, the conjunctiva is much more friable and the episclera much harder; the first tears easily and the latter is often annoyingly resistant to the needle, which may not be pushed too forcibly for fear of penetrating into the interior of the eyeball.

I overcome this difficulty in the following manner: if I think I have penetrated deep enough in the episclera, I carry the needle a little farther under the conjunctiva, and when the needle has come out at the point *b,* I introduce it again in order to seize a second fold of conjunctiva and give more firmness to the attachment of the muscle.

The threads being thus placed, warm sterile water is allowed to flow freely under the muscle and over the whole field of operation. The assistant then seizes the globe with a forceps on the side opposite the muscle to be advanced and turns it toward the latter, so that, in tying the threads, no traction shall be brought to bear upon the muscle nor upon the tissues included in the sutures. It is well to make first a simple knot for support by passing the thread twice around itself, first on one side and then on the other. One then finishes successively the two surgeon's knots. Finally the threads are cut short to prevent their ends from touching the cornea. We use one white thread and one black thread, the better to distinguish between them.

The operation completed, we once more flush the eye with sterilized water, and bandage both eyes. The dressing should be binocular even if but one eye has been operated upon, for does not even monocular strabotomy affect both eyes?[1] Does not the free eye, by its movements and by its accommodation, influence its fellow, covered or uncovered?

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[1] E. Landolt, L'opération du strabisme est toujours une opération binoculaire. International Medical Congress, Washington, D. C., 1887.

The occlusion of *both* eyes is intended to give them the immobility that is absolutely necessary to healing by first intention, a result indispensable in ocular surgery. The particular kind of motility of the globe, turning around a single center, makes it impossible to obtain this immobility by means of a compression bandage. Such a bandage might entail most serious consequences, especially after operations in which the globe is opened. The only means we have of immobilizing the eyes, at least relatively, is the removal, by simultaneous occlusion, of every reason for fixation and accommodation, and by keeping the patient as much as possible at rest. This condition is essential to obtaining a good result. If we close only one eye, if we allow children to be taken home after operation, or permit patients to have visitors in the hospital, we compromise the success of the most carefully performed operation.[1]

The patient must be quiet, in a well-ventilated room. He may have with him, if he so desires, some quiet person who may entertain him with music, or read to him an interesting but not exciting book. Our patients are visited by the chief of clinic morning and evening, and by me at midday. The dressing is usually changed every twenty-four hours, and the eyes are then thoroughly flushed with a warm, sterile, three per cent. solution of boric acid. In *convergent* concomitant strabismus, a drop of atropin is, at the same time, instilled into each eye. The sutures are removed on the fifth or sixth day.

In *divergent* strabismus, the eyes are left free from this time, for, accommodation and convergence acting in the same manner as the operation, can have only a favorable influence on its result.

In *convergent* strabismus, on the contrary, I prefer to allow the binocular bandage to remain a day longer, and when I remove it, I immediately replace it by convex glasses which correct the total hypermetropia, and, of course, any existing astigmatism. These lenses are of smoked glass, because of the mydriasis produced by the atropin.

The judicious use of the mydriatic and spectacles is an aid of great value in the hands of an intelligent observer, especially in convergent strabismus. So long as there is a tendency to excessive convergence, the suppression of accommodation will prevent the patient from falling back into his old infirmity. If, on the contrary, an overcorrection is feared,—which is very rare after muscular advancement,—first the mydriatic is discontinued, while the convex lenses are retained, at least for the correction of the manifest hypermetropia and for near work. If the latent divergence persists, let the spectacles be given up entirely, and one will be astonished at the rapidity with which the accommodation brings back the convergence.

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[1] E. Landolt, Congrès internat. des Sc. méd., Paris, 1900, Section d'Ophth., p. 289.

The best guide for the normal direction of the eyes and the correction of all strabismus, our most sure and most potent ally in all strabotomy, is, as I have always stated, *binocular vision.* All our efforts should tend to re-establish this important function.

I have often enough spoken of the means which we employ to this end; to revert to them would be to repeat myself. Let me only call to the attention of the readers my last stereoscope, which allows a variation in the amount of illumination of one of the images, so as to equalize the impressions received by the two eyes. It is very useful in re-establishing binocular vision.[1]

I might now describe briefly certain typical cases, in order to demonstrate the beneficial action of muscle advancement in *paralysis of the ocular muscles.*

Case I.—The first patient was a man, aged 53 years, who was attacked with *paresis of the left external rectus,* producing a convergent strabismus of 8° with homonymous diplopia. Our prisms are numbered according to the angle of deviation which they produce; so that a strabismus of 8° is corrected by prism No. 8, and that a correcting prism No. 8 indicates a deviation of the eye of 8°.

The nasal excursions of both eyes were normal; but, while the unaffected eye could turn 60° to the temporal side, the paretic eye (the left) stopped at 54°. The fixation field of this eye was therefore 6° less extended on the temporal side than the field of the unaffected eye.

I performed *advancement of the paretic external rectus* combined with a very slight *resection* of the tendinous extremity. As usual, both eyes were bandaged, the dressing being changed every twenty-four hours.

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[1] E. Landolt. Stereoskope, II. Internat. Congr. of Ophth., Utrecht, 1899, and Arch. d'Ophth., 1899.

Five days later the sutures were removed. Immediately after, there was still a divergence with 7° of heteronymous diplopia. This disappeared, however, the same evening, the patient spontaneously fusing the images of the two eyes.

One month after the operation all strabismus, all diplopia, had entirely disappeared and the amplitude of convergence even exceeded the normal.[1] The following result was obtained after the seventh day:

Maximum of convergence: $p = 12$ meter angles, instead of 10, which is the average.
Minimum of convergence: $r = -2$ meter angles, instead of -1, which is the average.
Amplitude of convergence: $a = 14$ meter angles, instead of 11, which is the average.

The great value of the *negative* part of convergence amplitude, of abduction or *facultative divergence,* shows how much the new insertion had improved the action of the external rectus. The great value of *positive convergence* on one side, the normal extent of *nasal excursion* on the other, proves that, notwithstanding the advancement of the external rectus, the internal rectus had lost nothing, either in the symmetrical movements or in the associated movements of both eyes.

The effect of a tenotomy of the internal rectus would have been precisely the reverse: Without any gain on the temporal side, the eye would have lost in excursion on the nasal side, as well as in amplitude of convergence. A divergent strabismus with crossed diplopia would have manifested itself in looking toward the side of the muscle which had been set back, but above all in looking at near objects.

Another function particularly interesting in this case was the *projection:* that is to say, the manner of locating an object seen with each eye alone, and with both eyes together.

I examine this function by means of an apparatus which I will describe at the end of this article.

In this case projection was very much altered. Instead of "projecting" toward the left (that is to say, to the side of the paretic muscle) objects which he fixed with the left eye alone, the patient localized well with this eye, and it was the unaffected eye, the right, which localized objects badly, and projected them to the right, when he fixed with this eye alone. This phenomenon, which is rather a rare one, may be explained by the fact that the visual acuity of the paretic eye was better than that of the other eye, which had a visual acuity of only 0.7. When, because of the paresis, the patient could no longer fix with both eyes simultaneously, he used preferably the eye which had the better vision, although this was the paretic one. He learned thus to localize correctly with this eye, and the false projection passed, so to speak, to the unaffected eye.[1]

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[1] E. Landolt, in Norris and Oliver, System of Diseases of the Eye, vol. iv, p. 129.

After the operation, projection became normal in both eyes; whether he fixed with the left eye alone or with the right, he localized perfectly the object looked at.

Case II.—The second patient was a confrère, about 40 years of age, who was also suffering from a *paresis of the external rectus of the left eye.* In this case, however, deviation and limitation of the field of excursion were much more marked than in the first case. He had a *convergent* strabismus of 18° with homonymous diplopia, and the temporal excursion of the affected eye was limited to 37°, that is to say, to 15° less than that of the unaffected eye, which reached 52°. In fixing with the left eye alone, the patient projected 11° to the *left.* Both eyes were emmetropic and their visual acuity was equal to 1.5.

The diplopia was extremely annoying, so much so that the patient readily accepted the proposed operation. This consisted, naturally, in *the advancement of the paretic external rectus* with *resection* of a more extensive portion than in the preceding case.

The sutures were removed on the fifth day after the operation. Fusion was immediately perfect, the amplitude of convergence was normal, and the temporal excursion of the eye operated upon reached 50° (instead of 37°, as before the operation). The patient was seen more than a year after the operation and the result had remained perfect.

Case III.—The third patient was a young man, 35 years old, with a *paralysis of the external rectus of the right eye,* which produced a *convergent* strabismus of 25° and a limitation of the temporal excursion of the eye to 8°. The corresponding excursion of the unaffected eye was 50°, the paralysis causing thus a loss of 42°.

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[1] Marc Landolt, Une particularité dans les symptômes de certaines paralysies oculaires. Arch. d'Ophth., xxii, p. 41, 1902.

The *advancement of the paralyzed muscle* was combined with a more extended resection than in the two preceding cases. Recovery was normal.

Seven days after the operation, there were still 13° of homonymous diplopia. The patient left the clinic, and practised the following orthoptic exercises: he placed himself at some distance from a light and turned his head to the right, until the images, at first separated, were merged into one. He then endeavored to maintain this fusion while turning his head gradually to the left, thus extending his field of binocular vision. Thanks to these exercises, and to the disappearance of the traumatic effects of the operation, binocular vision was re-established, not only in looking straight ahead, but even in looking toward the side of the paralyzed muscle. Homonymous diplopia was only manifest at the extreme right limit of the field of fixation.

When the patient returned four months later, both strabismus and diplopia had entirely disappeared.

The excursions of the right eye were 50° on the temporal side (instead of 8° !) and 50° on the nasal, equal to the excursions of the unaffected eye.

The amplitude of convergence was also normal:

The maximum, p = 11 meter angles.
The amplitude, a = 11.6 meter angles.
The minimum, r = —0.6 meter angles.

Case IV.—Case four was a particularly difficult one: a woman, 39 years old, attacked, in both eyes, with *paralysis of the sixth pair,* which produced a *convergent* strabismus and a *homonymous* diplopia of 30°.

The *temporal* excursion of the *left* eye was only 5°, and of the *right* only 8°. The *nasal* excursion reached 55° for each eye.

The patient had been accustomed to use her eyes without correcting-glasses, her visual acuity being 0.1 in the left, and 0.2 in the right. The correction of half a diopter of astigmatism increased the visual acuity to 0.3 in the left, and to 0.5 in the right, eye.

It was natural that the patient used almost exclusively her *right* eye, and that therefore this eye learned to locate accurately objects fixed, in spite of the paralysis of its external rectus. In fixing with this eye alone, the patient touched accurately with her right hand, and only 4° to the right with her left hand. On the contrary, in fixing with the *left* eye, she projected *more than* 25° to the *left.*

On November 3, 1900, I performed the *advancement of the external rectus of the left eye* with a *very extensive resection.* Seven days after the operation, there were but 8° of homonymous diplopia, instead of 30°.

The projection with the eye operated upon was correct when the patient used her left hand, the right hand still having a tendency to touch about 4° to the left of the object fixed.

When the patient fixed with the right eye alone, she made a mistake of 7° to the right with her left hand, and of 4° to the right with her right hand. In binocular vision, there was also a tendency to projection to the right, though less pronounced than before the operation.

On the 17th of November, I performed the same operation (*i.e., advancement and extensive resection of the external rectus*) on the *right* eye.

Seven days after the operation, there were still 6° of convergence with homonymous diplopia.

Projection with the left eye was 3° to the left, with the right eye 3° to the right.

Four months afterward the patient was still projecting 7° to the left when she fixed with the left eye alone, but correctly with the right eye alone, and in binocular fixation.

Fifteen days after the operation the patient was working binocularly without fatigue. One month later there was the same degree of convergent strabismus (6°), but diplopia no longer annoyed the patient, as she had learned to fuse the two images, by turning her head slightly to the left.

Less than three months after the last operation, diplopia had disappeared even in looking straight ahead, and only manifested itself in looking to the extreme right.

The excursions of the eyes were as follows:

The *left* eye:			
Temporal:	55°	instead of 5°;	difference +50°
Nasal:	45°	instead of 55°;	difference —10°
Together:	100°	instead of 60°;	difference +40°
The *right* eye:			
Temporal:	45°	instead of 8°;	difference +37°
Nasal:	40°	instead of 55°;	difference —15°
Together:	85°	instead of 63°;	difference +22°

In this case, the nasal excursions of the eye seem to have suffered somewhat in extent by the advancement, or, rather, by the resection, of the external recti. This is not to be wondered at. While the advancement of a muscle up to the margin of the cornea is not detrimental to the function of its antagonist, this cannot be the case when the change of insertion is complicated by such an extensive shortening as is required in *paralysis of both external recti.* But it should be observed that the eyes have gained much more in extent of excursion on the side of the advancement than they have lost on the opposite side; that is to say, the horizontal field of excursion has been increased for one eye 22°, and for the other 40°.

Tenotomy with a setting back of the internal recti in this case would have completely abolished the excursions to the nasal side, without correcting the convergent strabismus and without increasing the temporal excursions.

The combination of a tenotomy of the internal rectus with a simple advancement of the external rectus, performed upon both eyes, a procedure which would have been indispensable, might have remedied strabismus for distant vision, but the nasal excursions would have been diminished in much greater proportion than by our operation. There would have been divergent strabismus for near vision and a crossed diplopia quite as annoying for the patient as the homonymous diplopia which decided her to undergo the operation.

The effect of the double advancement without tenotomy of the antagonists, on the contrary, has been so satisfactory that the patient, whom I recently saw (nearly two years after the operation) is highly satisfied, and has assured me that she can work without fatigue with both eyes, which proves that, in spite of the slight diminution of the nasal excursions, convergence has remained sufficient.

Case V.—After these cases of paresis of the sixth pair, let me note one of *paresis of the common motor oculi:* A man, 36 years old, the left eye emmetropic with normal visual acuity, the right eye hypermetropic to 1.5D and with a visual acuity of but 0.3. Accommodation was impaired in both eyes, otherwise there was no evidence on the left side of alteration in the function of the third pair. Notably the excursions in the horizontal plane were normal, reaching 50° both on the temporal and the nasal sides.

The *right* eye, on the contrary, was attacked by *paresis* of the *entire common motor oculi.* There was *divergent strabismus* with 25° *of crossed diplopia,* and in direct vision this eye was 5° lower than its fellow. The difference in height varied necessarily with the gaze, the paretic eye falling behind the unaffected one in lowering as well as raising the gaze.

The symptom which most annoyed the patient was the great amount of crossed diplopia (25°). The right eye could turn inward only 30°, having therefore lost 20° on the *nasal* side. The patient projected, on an average, 15° to the *left* when he fixed with the *right* eye alone.

On December 27, 1899, I performed an *advancement of the internal rectus* of the *affected eye,* with a slight *resection* of the tendinous extremity.

Three weeks after the operation, the crossed diplopia had entirely disappeared. The difference in height only persisted (since we had not touched the muscle acting in the vertical direction), but the two images touched each other. These images fused completely as soon as we corrected the difference in level by the aid of prisms, combined with convex lenses which corrected the error of refraction. The patient has worked since with the greatest facility.

Case VI.—The sixth case is a very rare one: *isolated paresis of the superior rectus.* The young man, 28 years old, otherwise perfectly well, had been annoyed for more than five years by a *vertical diplopia* which had baffled the skill of the best specialists. One of these wished to entrust this interesting patient to me.

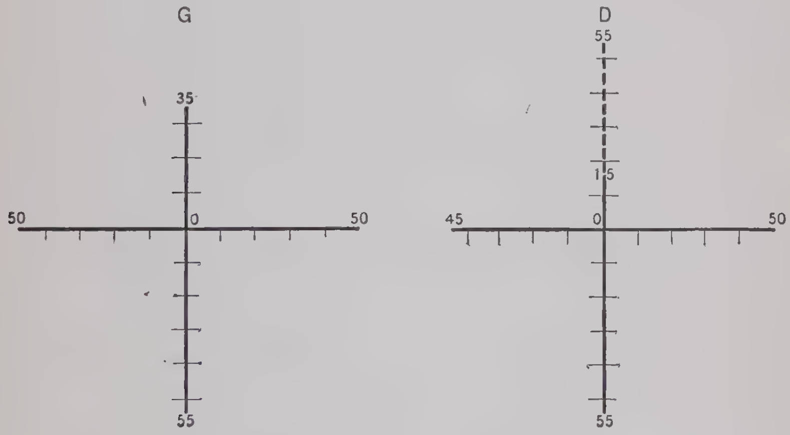
The characteristic symptoms left no doubt as to the diagnosis. There was *vertical* and *slight crossed diplopia* corresponding to a *lowering* of 13°, and a *divergence* of 3° in the *right* eye.

The excursions in the vertical and horizontal meridians are represented by the accompanying diagrams (Fig. 2), in which 0 corresponds to the point of intersection of the horizon with a line leaving each eye in a direction perpendicular to the face. The other numbers indicate, in degrees of excursion, the limits of the fixation fields in the horizontal and in the vertical meridians. They show for the *right* eye (the plain line) a diminution of 20° in *elevation,* and of 5° in the *nasal excursion,* in comparison with the left eye.

Advancement of the paretic superior rectus, with resection of its tendinous extremity, was done.

Five days after the operation, the sutures were removed, and on the seventh day the patient left Paris with a slight over-correction of 3°. This did not cause me any concern, for, although nothing is so much to be dreaded after a tenotomy as an over-correction, I consider a slight over-correction after muscle advancement in the light of a benefit. It is a sort of reserve force, which nature knows marvellously well how to dispose of for re-establishing the collaboration of the two eyes.

FIG. 2.



In fact, when I saw the patient 40 days after the operation, fusion was perfect, and the excursions in the vertical meridian were 55° *upward* and 50° *downward* in the eye which had been operated upon. (See dotted line in Fig. 2.)

I might describe, in order to show the excellent effect of muscle advancement, some cases of *paralysis of the superior oblique of a traumatic nature.* The paralysis was due to a radical operation upon the *frontal sinus.* But these observations having been already published by my pupil and friend, Dr. Stanculeanu,[1] I will limit myself to quote them in a few words.

In one of these cases, the traumatism had produced a condition similar to a paralysis of the fourth pair: a *strabismus sursum vergens of* 11°; and in the second case a *like strabismus of* 12°.

The superior oblique not being accessible to advancement, I performed that of the *inferior rectus.* In both cases, I had the satisfaction of re-establishing fusion in the entire lower part of the field of fixation without detriment to the upper part, and of giving to the patients, both of whom were working-men, the possibility of again taking up their occupations.

The few cases which I have described, as well as many others which I have subjected to the same measurements before and after operation (*degree of deviation* and of *diplopia, monocular* and *binocular projection, field of excursion, amplitude of convergence,* etc.), show that *muscle advancement* is the operation indicated in paralyses of the ocular muscles; that, well performed and properly treated afterward, this operation suffices to re-establish the normal function of ocular movements in the case of paresis of a slight or of an average amount. To this should be added the *resection* of a portion of the muscle *greater* or less in extent as the paresis is more or less marked.

By this method may be obtained a perfect field of excursion, binocular vision, and, a most important point, an *amplitude of convergence* which is excellent or at least sufficient in cases of paralysis of the sixth pair.

When paralysis is complete, the re-establishment of the excursion of the eye in the domain of the affected muscle is, of course, not possible: fusion could only be accomplished in direct vision and on the side opposite to the paralyzed muscle. When the gaze falls in the sphere of action of the paralyzed muscle, diplopia necessarily reappears, but it is scarcely annoying to the patient, since he may avoid it by slightly turning his head. At least one does not create a new diplopia in the opposite direction, as is the case when one endeavors to correct paralytic strabismus by means of tenotomy with or without advancement.

When advancement and extensive resection of a paralyzed muscle do not prove sufficient, I would have recourse to the advancement of the homonymous muscle of the other eye rather than to a setting back of its antagonist. Tenotomy, but a very delicate one, that is to say, subconjunctival and without detaching the muscle from its surrounding tissues, may be admissible in complete paralysis of long duration, when the antagonist has suffered changes in structure which prevent its relaxation, and when binocular vision does not exist because of the blindness of one eye.

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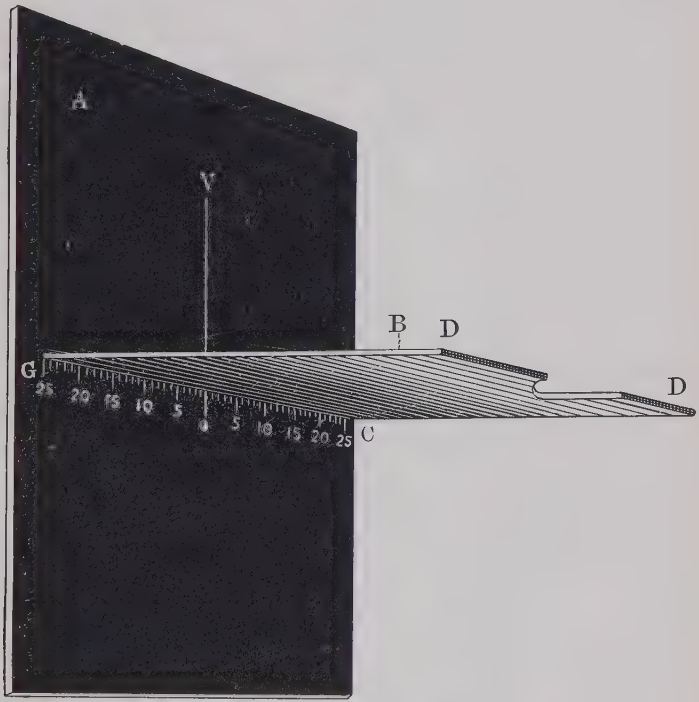
[1] Arch. d'Ophth., 1902, xxii, p. 32.

FIG. 3.



APPENDIX.—Following is the method by which I determine "projection,"[1] that is, the manner of locating an object fixed, or the spot where an object looked at is supposed to be situated.

A blackboard (A, Fig. 3), placed in an upright position, is crossed in the center by a vertical white line, V O. A smaller board, B, is fastened to it by means of hinges, G C. The other border,

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[1] E. Landolt, La détermination de la "Projection" ou "Localisation" de l'oeil, Arch. d'Ophth., Mai, p. 273, 1898.

D D, has a hollowed portion which fits the neck of the patient. During the examination the board, B, is placed horizontally.

The neck of the patient being well fitted in the curve, his eyes are seventy centimeters from the blackboard, and he sees the white line immediately in front of him above the small board, which entirely hides from him the lower part of the blackboard as well as his own arms.

Below the intersection of the blackboard with the small board is a scale, the zero point of which corresponds to the vertical line. To the right and left of this are indicated the tangents of the angles for a radius equal to the distance which separates the patient's eyes from the blackboard.

One eye of the person to be examined is covered, and I ask him to touch quickly, under the small board, the spot where he supposes the prolongation of the vertical line to be. Thus I note whether he projects correctly, and if not, in which direction and to how many degrees the patient is wrong. Since he does not see his finger nor even his arm, this observation is of much greater precision than when made without cover. But in order that the results may be depended upon, it must be done very rapidly, so that the patient may not have time to orient himself. It must also be done alternating with first one and then with the other hand, and the patient should not be told whether he touches correctly or not.

These observations have already given me very interesting results, often even quite unexpected ones, in cases of motor insufficiency, of concomitant and paralytic strabismus,—in showing, for example, that the eye quickly learns to modify its projection conformably to the necessity of orientation.

A paper that I published, in 1893, in the *Archives d'Ophthalmologie,* p. 257, upon the relation between the angle of primary and secondary strabismus, and the false projection, shows also the value of these investigations.[1] Other observations of even greater interest will be published later, when I shall have a sufficient number to allow me to draw definite conclusions.

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[1] E. Landolt, in Norris and Oliver's System of Diseases of the Eyes, iii, pp. 17, 18, and 19.

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